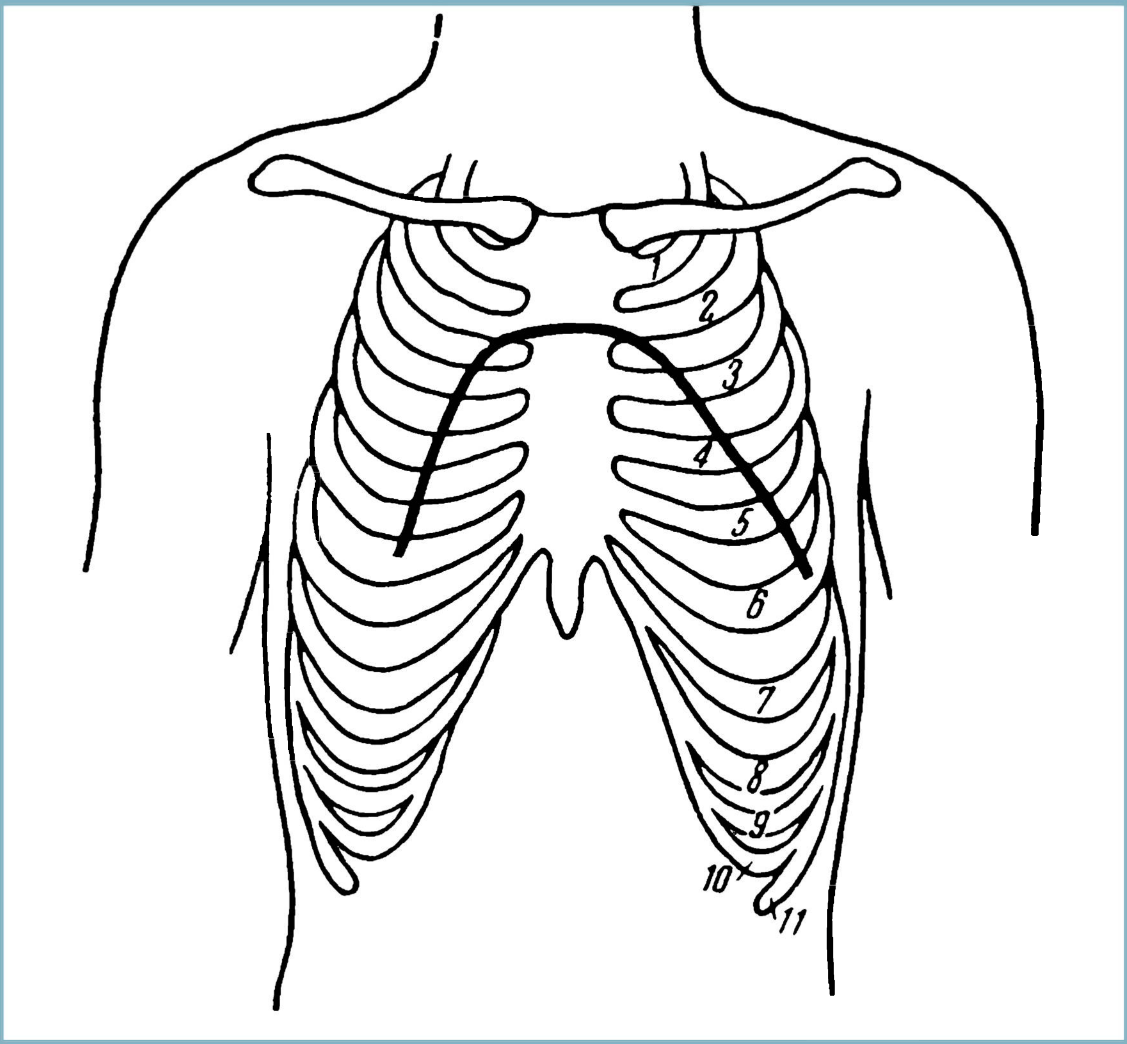
Aram GUKASYAN

Internal Diseases



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Internal Diseases

by

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An Introduction to Clinical Internal Medicine

THE SUBJECT OF INTERNAL MEDICINE

In the past, medical science was subdivided into internal medicine and surgery, while other specialities did not yet exist. Later, with the progress of sciences, a number of diseases—paediatric, nervous, skin and venereal, infectious, gynaecologic and others—were separated from internal medicine and became independent subjects of study.

Today, internal medicine deals with all diseases of the visceral organs requiring no surgical interference.

Internal diseases are the principal branch of medical learning which is indispensable for all medical workers of every speciality, since a disease may often be associated with a lesion of the visceral organs. For example, impairment of vision to the degree of total blindness is a major sign of chronic inflammation of the kidneys; eczema and other skin diseases are concomitant with diabetes mellitus; azotaemic uraemia is seen with compression of the ureters by a tumour, etc. It is clear, therefore, that a specialist in eye, uro-genital and other diseases should necessarily have the required knowledge of internal medicine.

CLASSIFICATION OF INTERNAL DISEASES

We talk about diseases of the stomach, kidneys, heart, etc. A lesion of any organ, however, is the result of a disease of the body as a whole. In inflammation of the kidneys, for example, not only the glomeruli and the tubular system of the kidneys but also the heart, the haemopoietic organs, and the nervous and vascular systems of the body become affected. Diabetes mellitus, in addition to a disturbance of carbohydrate metabolism, is manifested by a disorder in protein and fat metabolism. A disease of any visceral organ causes serious changes to occur throughout the body. Nevertheless, the classification of diseases has been drawn up so as to indicate in which organ these anatomical and functional changes are the most severe.

Recognition of the integrity of the body should by no means be interpreted in favour of renouncing the subdivision of medicine into separate specialities, because this would have a highly adverse effect on the standards of medical care of the public.

EXOGENOUS AND ENDOGENOUS CAUSES OF INTERNAL DISEASES

During his life, man is exposed to diverse external (exogenous) favourable and unfavourable influences. The latter are often responsible for various diseases. The external causes of a disease may be physical, mechanical, chemical and biological. Among the physical causes are chilling and overheating of the body, wholly or in part, heat and sun stroke, exposure to radium and x-rays, the action of electric current, lightning, and atmospheric pressure variations.

Mechanical causes are responsible for contusions, wounds, bone fractures, dislocations of joints, sprains, concussion of the brain, etc.

The chemical factors causing internal diseases include various poisons as well as war gases.

The biological causes of diseases are as follows: (a) disease-producing agents—microbes and viruses gaining entry from the external environment into the human body with air, water and food, through clothes, etc. They are responsible for influenza, tonsillitis, scarlet fever, diphtheria, dysentery, tuberculosis and other infectious diseases; (b) blood-sucking insects transmitting infection from sick to healthy persons. For example, lice are vectors of typhus and relapsing fever. Anopheles mosquitoes transmit malaria, fleas may spread plague, ticks, relapsing fever and encephalitis; (c) parasites—helminths and various fungi affecting the skin and even the visceral organs (actinomycosis); (d) malnutrition; partial or absolute fasting and hunger may be the cause of a disease and emaciation.

Another external cause of a disease is a harmful environmental influence on the central nervous system and the mental state of a patient. Constant anxiety, anguish of mind, and the so-called mental trauma may lead to various diseases of the heart, stomach, intestine, etc. A disease may also be provoked by the effect produced on a person by a spoken or written word. For example, news of the death of his near ones may make him develop a severe illness. Sometimes, even a word carelessly uttered in the presence of an ill person, especially one overanxious about his health will do him great harm.

A disease due to environmental influences, however, can develop only in persons with a reduced body resistance. Where the latter is sufficient, harmful environmental factors are powerless. For example, in food poisoning the ingested food is ejected from the stomach by vomiting. Foreign bodies are expelled from the throat by coughing; microbes invading the blood-stream may be destroyed by leucocytes, etc. This interaction between the environmental factors and the reaction of the body to them shows the latter's adaptational capacity, i.e., ability to neutralize harmful environmental influences and resist a disease. Body resistance depends on the condition of the central nervous system, as well as on hereditary and acquired properties.

A disease may also be due to an internal cause, either arising within the body or connected with characteristics inherited from parents or acquired during life.

PATHOGENESIS

Pathogenesis is a term denoting the routes by which a pathogenic agent gains entry into the body and the mechanism of the production of a disease. The condition of the central nervous system is a decisive factor in pathogenesis. The teaching on the critical role played by the central nervous system in the vital activity is known as nervism. It was founded by Sergei Botkin and further developed by Ivan Pavlov.

Pathogenesis also shows how various organs become affected in the course of a disease. For example, knowledge of the pathogenesis of a kidney inflammation helps understand the causes of the concomitant deterioration of vision and appearance of dyspeptic symptoms. Likewise, we can establish the cause responsible for heart failures, circulatory disturbances, changes in the kidneys and other morbid symptoms seen in rheumatism.

STAGES OF DISEASE

There is a certain interval between the time of entrance of a disease-producing agent into the body and the appearance of the initial and obvious symptoms of a disease. This interval varies with different diseases. In infectious jaundice it lasts 3-4 weeks, in scarlet fever 2 weeks, in chickenpox 3 weeks, etc.

The period of a disease between the implantation of the contagium into the body and the development of the initial symptoms is known as the incubation period. The first symptoms of a disease appear in the prodromal (precursory) period. These are malaise, general debility, partial or total loss of the appetite and sometimes a low-grade fever. It is difficult to identify a disease by these symptoms since they are not specific and precede the principal symptoms of diseases of variable aetiology. The prodromal period lasting several days is followed by the frank stage of the disease with its characteristic subjective and objective symptoms.

SUBJECTIVE AND OBJECTIVE SYMPTOMS OF DISEASE

A disease gives both subjective and objective symptoms. Examination of a patient is usually begun by asking him to give his complaints which make up the subjective picture of the disease and are a reflection or consequence of objective changes in the body. The objective picture of the disease is revealed by physical methods of examination, combined with clinico-laboratory, biochemical, and instrumental investigations (x-ray, electrocardiography, etc).

Special importance in the examination of a patient is attached to his complaints, which are taken down for a detailed study of the case history. It should be borne in mind that purely functional disorders may often be overlooked at physical examination, and are identified only from the patient's complaints. The diagnosis of a disease in its early stage is especially important in view of the absence at this time of severe and irreparable structural changes in organs, which fact accounts for the good effect of therapy undertaken in this period.

DIAGNOSIS AND DIFFERENTIAL DIAGNOSIS

Diagnosis consists in identifying a disease on the basis of the sufferer's complaints, history of the case and past diseases, life history and findings of physical examination. The diagnosis should take into account all aspects of the disease. First, the examiner determines the site of affection and the essence of the changes due to which the patient is applying for medical aid. Let us suppose a patient has been found to suffer from aortic valve insufficiency. The next stage in the diagnostic procedure will be to determine the aetiology, i.e., the cause of the above defect, because it is known to be of a variable origin—rheumatic, syphilitic or sclerotic. The third stage is a pathophysiological description of the defect, i.e., determination of the degree of circulatory disturbance.

A diagnosis may be difficult to make because identical subjective and objective symptoms are often found with different diseases. For example, gastric or intestinal haemorrhage occurs in ulcer, cancer, and polyposis of the stomach or intestine, liver cirrhosis, etc. A number of additional investigations, a study of the secretory function and x-raying of the stomach, etc., are used to rule out or establish the possibility of certain diseases. Identification of a disease in this way is known as differential diagnosis. An example in point is accumulation of fluid in the pleural cavity. This fluid may be of inflammatory or noninflammatory origin. The former in its turn may be sero-fibrinous, purulent, or haemorrhagic. The presence of inflammatory exudate may be due to a number of diseases: cancer, tuberculosis, lobar pneumonia. Fluid of a noninflammatory character, known as transudate, may be produced as a result of diseases of the heart, kidneys, etc. Examination of fluid obtained by a test puncture serves to determine whether it is inflammatory, while the cellular composition of the fluid residue often gives valuable diagnostic evidence. X-ray examination, as well as urine and blood analysis, also aid diagnosis.

When making a diagnosis, one should discriminate between the principal and the concomitant diseases. When two diseases are running a simultaneous course, the graver one is regarded as principal, the other one as concomitant. For example, in case of stomach cancer concurrent with chronic bronchitis, the former is the principal disease, the latter the concomitant one.

THE COURSE OF INTERNAL DISEASES

Diseases may be acute or chronic, depending on the duration of their course. The former are of a sudden onset, and after a brief period (6-8 weeks) end in recovery or sometimes in death. The latter diseases run a prolonged course, with periodic aggravations. Both acute and chronic diseases are often accompanied by complications. For example, lobar pneumonia, an acute disease, may become complicated by pleuritis. A lung abscess, which runs a chronic course, may be complicated with pleural empyema, etc.

A disease sometimes causes temporary or permanent incapacity for work.

A number of chronic diseases culminate in permanent disablement, which may be partial or total.

PROPHYLAXIS AND TREATMENT OF INTERNAL DISEASES

Prophylaxis of disease is the fundamental principle of the Soviet health service which is based on an all-round development of preventive medicine as the basic factor contributing to a reduction of morbidity and mortality and to a radical promotion of public health. Prophylaxis may be collective or individual. Collective prophylaxis consists in government measures of epidemic control, improvement of the working and living conditions of the public, checking the quality of foodstuffs at public catering establishments, etc. Collective prophylaxis is also carried out by setting up holiday homes, lying out children's play-grounds, opening boarding-schools in the countryside, widely promoting physical culture and sports, undertaking vaccinations against infectious diseases. A tremendous role is played by a free clinico-prophylactic service to wide sections of the population for detecting the initial symptoms of hypertensive vascular disease, malignant newgrowths, tuberculosis, and other diseases. Individual prophylaxis consists in observance of the rules of personal hygiene. An important place in the treatment of diseases belongs to medicinal substances, which should be applied in combination with general hygienic measures strengthening body resistance. That is why it is unwise to restrict medical treatment to the use of medicaments alone. Whenever possible one should resort to all remedies which effectively invigorate the body fighting a disease.

Pharmacotherapy may be aetiological, pathogenetic, or symptomatic. Aetiological therapy is meant to destroy the cause of a disease. For example, in syphilis aetiological therapy is with novarsenol, in malaria with quinine, etc. Pathogenetic therapy is aimed at repairing the disturbed bodily functions. For example, in lobar pneumonia, in addition to aetiological therapy, strychnine is required to raise the vascular tone declined as a result of general intoxication. In this case, the application of strychnine is what we call pathogenetic therapy. In some cases, it is necessary to undertake also symptomatic therapy which is aimed at abating especially obnoxious disease symptoms. For example, pain is relieved with revulsive remedies or narcotics, a dry and painful cough with dionine or codeine, and so forth. Pathogenetic and symptomatic therapy is particularly important where the aetiology of a disease is unknown. In such a case, the functions of the affected organs are improved by means of drug treatment, dietetic therapy, and climatotherapy.

A combination of several therapeutic measures is known as combined therapy. This includes, among other things, dietetic therapy which can be effective against emaciation, obesity, and circulatory, metabolic, gastro-intestinal and other disorders.

Medical care should be based on the principle of individual approach taking into consideration all specific traits of a particular patient.

Great importance in the therapy and prophylaxis of diseases is attached to the use of natural therapeutic factors. A locality with a salubrious climate and other wholesome physical characteristics is often used as a health resort. Mineral springs yield water which may contain gases (hydrogen sulphide, carbon dioxide), radon, silica, magnesium, sulphates, iron, common salt, iodine, etc. The therapeutic effect of mud is due to its rich content of organic matter produced by bacteria. Another important health-promoting factor is the sea, which mitigates the littoral climate and contains various salts and organic substances dissolved in its water.

The climate is a highly important factor in the treatment of disease. It varies in type from that of the seaside to that of the mountainous and steppe regions. The climate of the woodland with its wholesome air is also very salubrious. The site of a climatotherapeutic centre is usually chosen in accordance with the annual number of sunny days, mean temperature and humidity of the air, and wind speeds in a given locality.

Treatment using some of the sources of nature, for instance, muds and mineral water, may also be applied at a patient's permanent residence.

Treatment at health resorts is mostly prescribed for chronic diseases; however, in certain diseases it is contraindicated. Therefore, treatment at a health resort should be prescribed not only for the principal but also the concomitant disease. For example, mud cure is helpful in inflammations of the female genitalia, but when the latter occur in combination with lung tuberculosis, even if abated, this treatment will be harmful since it may revive the tuberculous process.

Combined therapy is widely applied at health resorts and sanatoria where sun, air, sea, and mineral water baths as well as dietotherapy are employed in addition to physiotherapy (quartz and Sollux lamps, diathermy). The physical and balneological methods of treatment consist in simulating the nerve-endings in the human skin so as to provoke a change in the metabolic processes. Very important for rest and recuperation is the change of a patient's habitual environment, in particular, the beautiful scenery of a health resort.

Psychotherapy and control of iatrogenic diseases. In Russia treatment by suggestion had been given great attention since the time of M. Mudrov, but it was not before I. Pavlov developed his theories that psychotherapy was put on a scientific foundation.

It should be kept in mind that sometimes a word carelessly dropped by a medical worker may be responsible for a disease. There is even a group of diseases called iatrogenic, i.e., generated by a physician (from Greek *iatros*—physician). Every medical worker should display great caution toward his patients. For the sake of psychotherapy medical personnel are obliged to set up close contacts with the patients, win their confidence, which is possible only on the basis of mutual respect. The quiet surroundings and strict regimen largely contribute to recovery. A cozy ward in which no noise nor loud talk is allowed and the squeaking of doors and other irritating environmental influences have been eliminated serves to ease nervous strain, and promotes recovery.

THE HISTORY OF INTERNAL MEDICINE IN RUSSIA

Soviet medicine with its prophylactic principles, progressive world outlook and lofty humanitarianism has developed to its present thriving state on the basis of the teachings of Russian great men of science. Soviet medicine owes many of its great achievements to the progressive views held by outstanding Russian medical men. A prominent place among them belongs to the founders of Russian therapeutics, M. Mudrov, S. Botkin, G. Zakharyin, A. Ostroumov, V. Obraztsov, M. Konchalovsky, N. Strazhesko, and G. Lang.

Matvei Mudrov (1776-1831) laid the foundation of Russian medical science. He was Russia's first medical man to introduce case records into clinical practice and draw up a clinical examination scheme. He also improved the methods of objective study. His contributions in the field of therapy are equally great. He advised an individual approach to treatment, i.e., one taking into consideration all characters of a particular patient. Without restricting himself to drug therapy he tried to use the curative sources of nature, physiotherapy, dietetics, and physical exercise; he attributed special importance to hygienic habits and mental calm of the patient and to psychotherapy making the patient believe in his recovery. Mudrov gave an exceptionally profound description of the importance of prophylaxis and hygiene since in his view diseases were associated with unfavourable environmental conditions. He used to say that to take care of healthy persons and protect them against hereditary and other formidable diseases, as well as to advise them to lead a healthful life was "honest and unburdensome for a physician because it is easier to prevent than to cure a disease". Mudrov died of cholera in St. Petersburg in 1831 when he was taking part in epidemic control work.

Grigory Zakharyin (1829-1897) was an outstanding clinician, a talented scientist and pedagogue. He became famous for his original method of history taking by a thorough study of the patient's living conditions and manner of life and by detection of the causes of his disease. He also identified functional disorders in individual organs by studying the patient's complaints. Zakharyin did much to promote the introduction of methods of objective study into clinical practice. In the field of clinical medicine he worked to develop the hygienic and prophylactic principles. He regarded the amelioration of infavourable environmental conditions as the most important measure of public health promotion. He wrote that only hygiene is capable of an effective control of a mass disease.

Zakharyin was the first to point out the need for hygienic conditions at schools and the use of forces of nature for children's health promotion. He also took a stand in favour of balneotherapy and climatotherapy, scientifically substantiated indications and contraindications for mineral water therapy, and worked out methods for its procedures and dosage. He advocated the development of resorts and recommended the rural climate as a remedy against disease. Zakharyin continued work in reforming the system of higher medical education, which had been started by Mudrov and other eminent Russian medical men and helped to set up specialized clinics of pediatric, gynecologic, as well as skin and venereal diseases.

G. Zakharyin has left a rich scientific legacy. He drew up a classification of lung lesions in tuberculosis, was the first to describe the clinical aspects of heart and lung syphilis, published a new method for treatment of liver cirrhosis with calomel (mercurous chloride), expressed himself in favour of an infectious origin of cholelithiasis, and corroborated the usefulness of blood-letting by scientific evidence. The clinical lectures he published are still very popular among members of the medical profession.

Zakharyin's great merit is also the attention he gave to the importance of the nervous system in the vital functions of the body.

Sergei Botkin (1832-1889) made a big contribution to Russian science with his progressive ideas, and was known as an advocate of the physiological and hygienic principles in medicine. Botkin regarded the human body as being interrelated with the external environment while diseases and health were, in his view, the result of environmental influences upon the body. "The concept of a disese," he writes, "is inseparably linked with its cause, which is always conditional upon the external environment influencing a sick person either directly or through the agency of his near and distant ancestors."

S. Botkin noted the capacity of the body to adapt itself to changing environmental conditions, fortify the acquired qualities and hand them down to posterity.

His materialistic understanding of the integrity of the body and its unity with the external environment made him capable of a new approach to the solution of another important problem of medicine—that of pathogenesis. He gave the answer to this problem in his theory of nervism based, as it was, on a number of I. Sechenov's physiological discoveries. Botkin believed that external agents produced their pathogenic effect on the body through the peripheral nerve endings, from which the stimulation was conveyed to the central nervous system as impulses. His teaching on the leading role played by the nervous system in the development of a disease laid down the foundations for a new trend in medicine, known as the physiologic school.

S. Botkin attached great importance to the laboratory methods of investigation. Even during the first year of his work at his clinic he set up a research laboratory. Workers of this laboratory, in addition to clinical analyses, experimented with administration of medicines to animals. Many of these studies carried out under the general direction of Botkin were important events in the history of Russian medicine. Among them was a research into the influence of Adonis vernalis, digitalis, and convallaria majalis on the blood circulation.

S. P. Botkin is the founder of military therapeutics. In his papers he noted features peculiar to the course run by certain internal diseases under the conditions of active warfare, and was the first to describe a new disease to which he gave the name of Volhynia (trench) fever. He also worked out the basic principles of organization of therapeutic service in the Army and secured the enforcement of quinine prophylaxis of malaria in the Russian Army. Botkin outlined the duties of army therapeutists, urging the need for their full competence in the field of infectious diseases, prophylactic medicine and sanitary measures so important in wartime.

Botkin is also known for his great work in reforming the system of medical training. He insisted that women should be allowed to study medicine and was the first to have admitted a woman to the staff of his clinic. She was V. Kashevarova-Rudneva, the first woman ever to receive her M. D. degree in Russia. He is also credited with the setting up of the Society of Sisters of Charity which trained nurses.

Alexei Ostroumov (1844-1908) is the author of several valuable monographs on experimental physiology describing the important role played by the nervous system in perspiration, dilatation and constriction of vessels, etc.

He devoted much attention to problems of heredity and constitution. In the controversy over heredity he adhered to the view that newly acquired qualities and characters were inheritable. He maintained that organs of the human body became weaker or stronger during interaction with the external environment. Ostroumov considered it possible to improve the health of persons susceptible to disease in an unfavourable environment by changing their living conditions. That is why he demanded that better subsistence should be provided to those liable to contract disease because of their inadequate adaptation. His views of the human constitution were also progressive. He asserted, for example, that an asthenic body-build and a tower skull may be encountered in perfectly normal people and, conversely, people with a normal body-build may be liable to disease.

Ostroumov proved that in many diseases there is no conformity between the degree of morphological changes and functional disturbances, because slight morphological changes are often seen with severe functional disturbances, and vice versa.

He worked out the principles of the reflex or reflected functional disturbances in organs. In his view, the integrity of the body and the functional relationships between individual organs are effected through the nervous system, therefore, cholelithiasis is often accompanied by an attack of angina pectoris, and nephrolithiasis by dyspepsia and vomiting. He attributed much importance to the central nervous system and trophic influences in the development of functional disorders.

Vasily Obraztsov (1852-1921) became famous as a scientist and clinician. He developed an original method for examination of organs of the abdominal cavity—a methodical, deep sliding palpation to ascertain accurately the topography of the abdominal organs and the changes therein. He introduced into clinical practice methods of one-finger flick percussion as well as the methods for immediate auscultation of the heart, i.e., by applying the ear directly to the chest.

World medicine is indebted to V. Obraztsov and his disciple N. Strazhesko for their description of the clinical aspects of myocardial infarction, which has considerably facilitated the diagnosis of this widespread disease.

Obraztsov gave much attention to the training of research personnel and lecturers in medicine and founded a brilliant school of followers. Among his disciples N. Strazhesko was particularly remarkable.

Soviet medicine has made great progress owing to the Socialist system. The growing welfare of the Soviet people, organization of new medical institutions, scientific research centres and new higher medical schools have helped to undertake a large-scale campaign against epidemics and various diseases and an all-round introduction of prophylactic and hygienic measures.

The leading role in this noble work was played by the outstanding Soviet therapeutists M. Konchalovsky, N. Strazhesko, and G. Lang, who, with the assistance of their followers, laid the foundations of Soviet medicine and enriched it with many important discoveries.

Maxim Konchalovsky (1875-1942) supported the physiological trend in clinical medicine. He taught that body reactivity and its adaptational capacity is of great importance for the origin and course of a disease. He considered the disease as being connected with the functional condition of the body as a whole. Konchalovsky is the author of the teaching on the premorbid conditions (i.e., conditions preceding a disease). He asserted that to eradicate severe diseases responsible for disablement and a high mortality rate one should fight what at first glance seem to be innocent diseases from which they take their origin. Disease incidence and mortality can be considerably reduced by effective measures against chronic inflammatory processes in the bronchi and lungs, which prepare the ground for lung cancer, against tonsillitis entailing rheumatism or nephritis, against secretory neuroses of the stomach ending in gastric ulcer.

Konchalovsky did much to promote work in rheumatic fever control. He is the author of the infectious-allergic theory of the aetiology of rheumatism according to which body reactivity plays a decisive role in the origination of this disease.

He was President of the U.S.S.R. Committee for Rheumatism Research and Control and Vice-President of the International Antirheumatic League.

Konchalovsky has contributed a new chapter on rehabilitation prognosis in the field of internal medicine. He is also known for his studies in the field of haematology and blood transfusion. His views of diagnosis were quite original. Confident that the pathology of the body is systemic in character he recommended a diagnosis based on syndromes rather than on organopathological symptoms since the former reflect the regularity of systemic lesions of organs and therefore give more detailed information on the essence of pathology.

Nikolai Strazhesko (1876-1952) was an outstanding clinician and scientist. He had a remarkable keenness of observation and worked a lot to improve the methods for examining the visceral organs, in particular those of the cardiovascular system and gastrointestinal tract.

Strazhesko described the so-called cannon-ball sound heard in heart-block, as well as many important symptoms in diseases of the gastrointestinal tract. His studies on angina pectoris, rheumatism, and bronchial asthma based, as they are, on the principles of the physiological theory, are an important contribution to Soviet medicine.

He showed much interest in biochemistry and pathophysiology, as is evidenced, in particular, by his elaborate study of the problem of cardiovascular insufficiency. His studies on sepsis and focal infections, fatigue, longevity, and wound sepsis, based on a wide physiological foundation and combining clinical observations with morphological, microbiological, and biochemical investigations, have proved of great value.

All of his studies show his understanding of the importance of the physiological trend in medicine, as was demonstrated way back in 1901 in his dissertation on the "Physiology of the Intestine" and experimental research into the influence of bitter drugs on the secretory function of the gastric glands. His monograph "Foundations of Physical Diagnosis of Diseases of the Abdominal Cavity" is a handbook for Soviet therapeutists.

Georgy Lang (1875-1948) is known for his studies on functional pathophysiology. He invariably gave much attention to research in the regulation of vital processes, particularly in the field of cardiovascular pathology. Special importance is attached to his studies on hypertensive vascular disease in which he proves its neurogenic origin. His monograph "Hypertensive Disease" published posthumously was awarded a State Prize in 1950. G. Lang drew up a classification of cardiovascular diseases and described the essence of myocardial dystrophy which he attributed to the chemical and trophic changes in the heart muscle due to adiposity, dysthyreosis, anaemia, etc.

He has made a large contribution to the study of cardiac fibrillation and neurohumoral regulation of the blood circulation. He also studied diseases of the blood. In opposition to the morphological school in haematology, Lang developed a new functional theory which considers diseases of the blood as being associated with biochemical metabolic processes as well as disturbances in the nervous regulation of blood circulation and destruction of blood cells. G. Lang is also known as a capable lecturer.

He was an active leader of the Leningrad Therapeutic Society and Chairman of the Board of the U.S.S.R. Society of Therapeutists.

PAVLOVIAN PHYSIOLOGY AS THE SCIENTIFIC BASIS OF SOVIET MEDICINE

Nervism, advanced by I. M. Sechenov and worked out in detail by S. Botkin, was the principal idea running through the lifework of I. Pavlov and widely developed in his studies. He regarded nervism as a school of physiology trying to trace the influence of the nervous system to as many functions of the body as possible.

Pavlov has proved that the functions of the body are based on the conditioned reflex activity of the central nervous system.

The theory of nervism also substantiates the integrity of the body which is ensured by the nervous system, the principal regulator of all bodily functions. Man's existence is inseparably bound up with the external environment, including the social environment. Man's unity with the external environment and behaviour are conditioned by stimulations, sensations and impressions received through the organs of sense. Animals receive their impressions of the external environment only by direct stimulation, through the first signal system, whereas a human being, in addition to this route, receives impressions and sensations also through a spoken or written word. This is known as the second signal system which is characteristic exclusively of man. Pavlov's teaching on the second signal system is of tremendous importance. A word may provoke a disease in a human being, but it may also be a powerful factor promoting recovery.

Ivan Pavlov's physiologic theory is the scientific basis of Soviet medicine.

Examination of the Patient

Before treatment of a disease it is necessary to make a diagnosis (Gr. *diagnosis*—identification of disease), determine its aetiology (Gr. *aetia*—cause), pathogenesis (Gr. *pathos*—suffering, disease; Gr. *genesis*—origin), i.e., the mode and mechanism of its development, as well as the symptoms (Gr. *symptoma*—sign) by which it manifests itself.

The branch of medicine dealing with the methods for identifying diseases is known as diagnostics. A number of different procedures are used to establish a diagnosis: history-taking, physical examination (visual inspection, palpation, percussion, auscultation), laboratory studies (urine, faeces, blood, sputum and other analyses), instrumental studies (electrocardiography, rectoromanoscopy, cystoscopy, etc.), x-ray examination, etc. Very important for identifying a disease is knowledge of its symptoms (dyspnoea, oedema, jaundice, cough, fever, diarrhoea, vomiting, haemorrhage, headache, dizziness, general debility, etc.). Some of these symptoms are objective (diarrhoea, vomiting, haemorrhage), because they are identified by objective study, while others (headache, dizziness, debility) are subjective since they are evident only to the patient.

One symptom, however, is not enough for identifying a disease because identical symptoms are given by a variety of diseases. For example, enlargement of the liver may indicate a disease of the latter, as well as malaria or heart disease. Haematemesis (vomiting of blood) is encountered in ulcer, cancer, and polyposis of the stomach, liver cirrhosis, etc. Haemoptysis (expectoration of blood) occurs with various lung diseases (cancer, tuberculosis, bronchiectasis, infarction) and with mitral stenosis. Different diseases may give identical subjective symptoms, for example, pain in the heart region felt in myocardial infarction, pericarditis, certain heart defects, etc.

A disease can be identified only after detailed information has been gained on its origin, development and symptoms and the patient's medical history. The diagnosis requires complete and systematic collection of all memoranda and notes of a case according to a definite plan.

The great men of Russian medicine have made an important contribution to the existing methods for examining the patient. M. Mudrov was the first Russian clinician who attached great importance to the patient's complaints and information about his living conditions. Later, G. Zakharyin took special note of the patient's account of his life and development, as well as his surroundings. The methods for examining the patient worked out by Zakharyin were made more elaborate by S. Botkin, who showed special interest in the central nervous system and functional changes in bodily organs. A. Ostroumov supplemented these methods with questions concerning heredity and constitution and with a functional examination of organs. Later, V. Obraztsov proposed his procedure of methodical, deep, sliding palpation of the abdominal organs.

The plan of a patient's medical examination consists of the following sections: (1) an account of complaints, evidence on the present and past diseases, a history of life, sexual and family life, in particular, heredity; all such information is gained by questioning the patient or his relatives; (2) data of objective examination; (3) notes on the course of a disease (diary), i.e., records of daily observation of the patient's condition and individual symptoms. The aggregate of all these data as well as the epicrisis containing the physician's conclusions concerning a given disease makes up the case history.

HISTORY-TAKING

The patient's complaints. The patient's complaints are a source of information on his sensations and feelings, i.e., the subjective symptoms of a disease. The patient's description of his sensations with indication of their duration, place and time of occurrence as well as the circumstances provoking or obliterating them gives one a certain idea of the character of his disease. For example, complaints of a brief pain in the heart region after excitement or physical strain, radiating to the left shoulder-blade and arm and accompanied by numbness of the fingers may suggest angina pectoris. Complaints of pain in the epigastrium felt 1-2 hours after a meal, on an empty stomach or at night, abating after drinking milk or eating dry bread may give reason to suspect duodenal ulcer, etc. A patient, however, is often unable to give a coherent account of his complaints, or the latter are very uncertain. In such cases, the physician should take the initiative and specify the character of such complaints by asking additional questions. During an examination, however, a patient should not be asked questions likely to prompt a positive or negative answer misleading with regard to his condition.

The next step after collection of complaints is the record of anamnesis, which consists of two parts: information on a given disease and information on past life.

Anamnesis of a disease (Gr. *anamnesis*—a recalling). During history-taking, a patient may give so systematic and complete information on his disease as to enable the physician to undertake objective examination immediately. In most cases, however, one has to put additional questions to ascertain the character, cause and course of a disease. Such questions refer to: the onset of a disease (sudden, gradual), the time and site of its first symptoms, its causes (in the patient's opinion), the conditions and surroundings in which it began, the course (progression or amelioration of symptoms), treatment undertaken (which drugs or other remedies have been used).

Additional questions may help gain valuable information aiding diagnosis. For example, the diagnosis of pulmonary tuberculosis is largely facilitated by the patient's account of contact with tuberculotics. If a disease is preceded by tonsillitis, chilling, or influenza one will be justified in suspecting pneumonia, an acute inflammation of the kidneys, rheumatism, etc.

A patient's indication of his taking antipyretics may suggest the cause of variations in his body temperature. When collecting complaints concerned with a given disease, one should ascertain the patient's general condition (degree of debility, emaciation, the condition of the respiratory, circulatory, and alimentary organs, urine secretion, as well as the state of the nervous system).

Anamnesis of past life. The next step after hearing the patient's complaints is to collect information on his life in the past, known as anamnesis vitae (*vita*—life), according to the following plan: the patient's biography, diseases sustained, working and living conditions, bad habits, intoxication, sexual and family life, heredity.

Essential items in the biography are the age, birthplace and domicile. This information is especially important in case the birthplace and domicile are in regions with a high incidence of certain diseases (endemic goitre, opisthorchiasis, amoebic dysentery, brucellosis, etc.). Other important facts to make clear are the welfare and housing standards as well as the moral situation at all times of life, in childhood and adolescence in particular, when the body is especially susceptible to environmental influences.

The patient should be told to give an account of all diseases he has ever sustained, with special emphasis on whether or not they were followed by complications. This information is important in view of the fact that some diseases leave immunity upon recovery, for example, typhus, children's infections, while others, such as tonsillitis and influenza, are likely to cause serious complications in the kidneys, heart and joints. If a patient is unable to tell whether he has sustained a particular disease because he often does not know its name, he should be asked to describe its symptoms. For example, repeated chills attended with a high-grade fever and followed by profuse perspiration are characteristic of subacute bacterial endocarditis, copious discharge of sputum indicates a suppurative process in the lungs, etc.

After this one should find out about the patient's harmful habits, working conditions, occupational hazards (lead, mercury, dust, etc.) and intoxications to which he is exposed, special attention being paid to his possible abuse of alcoholic drinks, tobacco and narcotics, since they are liable to provoke chronic neurovascular and other diseases. It is also important to find out about the patient's housing and living conditions, whether or not he leads a healthful life during his holidays, goes in for sports, etc.

Abnormal sexual life and an unfavourable family situation may adversely affect a patient's neuropsychic sphere and cause a disease. That is why it is necessary to find out about the patient's sexual life and its normalcy. The condition of the genital function in a male, the time of the onset of the menses and their regularity in a female, pregnancy, childbirth, artificial and spontaneous abortion, stillbirth, the time of the menopause (cessation of menstrual function) and the pathologic symptoms attendant on it (blood flushes to the head, palpitation, increased excitability, excessive perspiration) should all be taken into account during examination of a patient.

History-taking is rounded off by ascertaining the state of health and life-span of the nearest and, in so far as practicable, distant relatives. It should be established whether there have been cases of syphilis, nervous and mental diseases, lung tuberculosis, alcoholism, diseases due to metabolic disorders (obesity, diabetes mellitus, etc.) among the patient's relatives.

Information about heredity is important because it may give a clue to the patient's predisposition to one disease or another. This predisposition should be understood as the result of environmental influences on the preceding generations which have contributed to the development of definite characters inheritable by progeny. Hereditary transmission of unfavourable characters, however, does not mean transmission of a disease. This is only confirmation of predisposition which may lead to a disease when corresponding conditions are available. Inherited predisposition may be offset by appropriate prophylactic measures.

It should be borne in mind that identical diseases occurring in members of one and the same family, for example, hypertensive disease, gastric ulcer, bronchial asthma, and obesity, may often be due to identical living conditions.

OBJECTIVE EXAMINATION

The next step after collecting anamnesis, i.e., information on the past history of a case, is to determine the patient's present condition—status praesens—by means of the following physical methods of examination: (a) visual inspection; (b) palpation; (c) percussion; (d) auscultation. During these procedures the examiner is guided by superficial landmarks on the body. These are as follows: on the thorax: (1) the anterior median line (linea mediana anterior) passing along the middle of the sternum and further downwards through the navel; (2) the right and left sternal lines (linea sternalis dextra et sinistra) running along the edges of the sternum; (3) the right and left midclavicular lines (linea medioclavicularis) passing through the middle of the clavicle; (4) the right and left parasternal lines passing between the sternal and midclavicular lines; (5) the anterior, median and posterior axillary lines passing vertically through the anterior, median and posterior edges of the armpit; (6) the right and left scapular lines (linea scapularis dextra et sinistra) passing through the inferior angle of the scapula when the shoulder is lowered; (7) the paravertebral line (linea paravertebralis) passing between the vertebral and scapular lines; (8) the vertebral line (linea vertebralis) running over the spinous processes of the vertebral column (Figs. 1, 2).

For orientation on the thorax it is necessary to note the rib or vertebra at the level of which a change has been found in each particular case. It

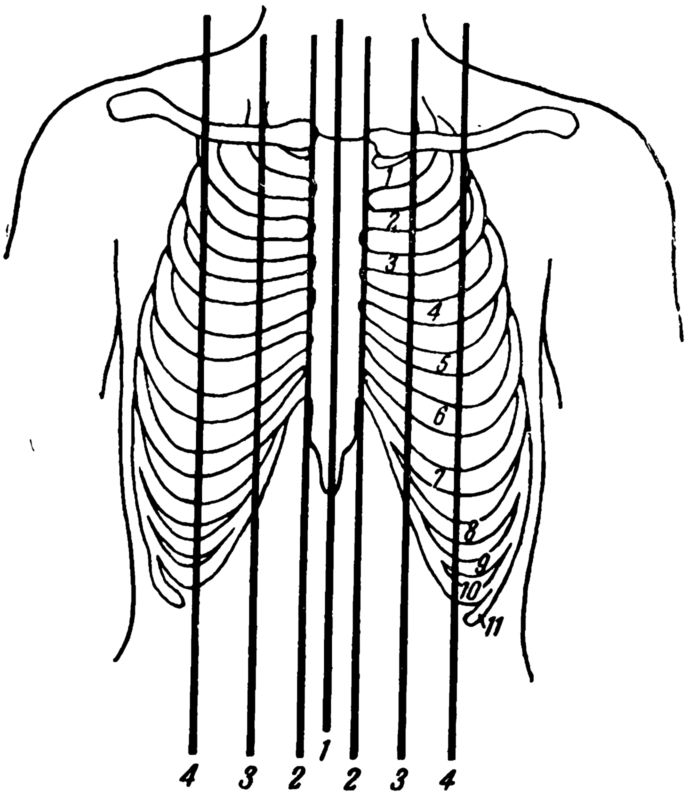


Fig. 1. Topographic lines on the anterior surface of the thorax

1—median line; 2—sternal line; 3—parasternal line; 4—midclavicular line

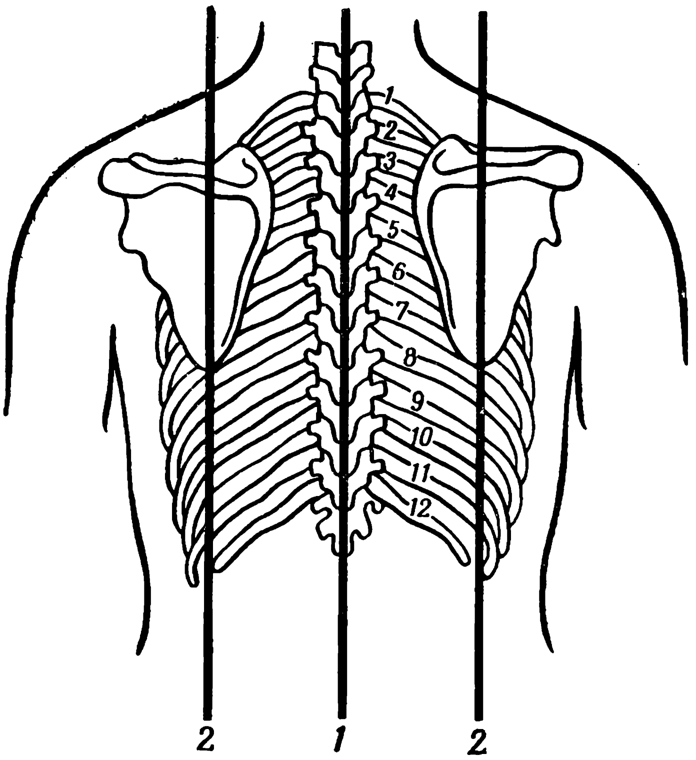


Fig. 2. Topographic lines of the posterior surface of the thorax

1—vertebral line; 2—scapular line

should be borne in mind that the first rib is covered by the clavicle and the second rib is united with the sternum at the site of attachment of the latter's manubrium and body.

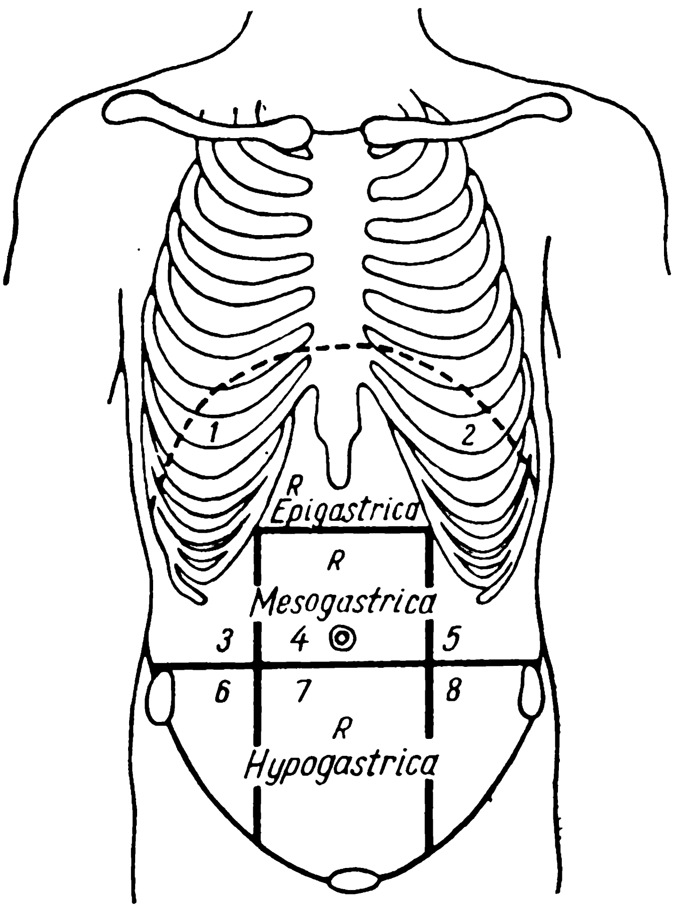


Fig. 3. Topographic regions of the abdomen

The spinous processes are counted from the most prominent seventh spinal cervical vertebra (vertebra prominens). The shoulder-blades occupy the space between the second and the seventh ribs. There are two fossae—the supraclavicular and the infraclavicular—above and under the clavicle, respectively. The abdomen is also subdivided into several sections (Fig. 3). Two imaginary horizontal lines divide the abdomen into three parts. The upper horizontal line connects the lower ends of the tenth ribs, the lower horizontal line connects the superior anterior spines of the iliac bones. The part of the abdomen above the upper horizontal line is called the epigastrium (regio epigastrica), the middle part of the abdomen (regio mesogastrica) lies between the two horizontal lines, and the hypogastrium (regio hypogastrica) below the lower horizontal line.

Two vertical lines running along the external borders of the rectus abdominis muscles divide the above-mentioned three parts of the abdomen into the following nine regions: (1) the right hypochondriac (regio hypochondria dextra); (2) the left hypochondriac (regio hypochondria sinistra); (3) the epigastric region (subscrobiculo cordis) between them. The middle part of the stomach is subdivided into two lateral regions—the right and the left (regio abdominalis dextra et sinistra) with the umbilical region (regio umbilicalis) between them. On the sides of the lower abdominal region there are (a) the right and (b) the left inguinal regions (regio inguinalis dextra et sinistra), and the suprapubic region (regio suprapubica) between them.

EXAMINATION OF THE PATIENT

Examination of the patient may yield valuable information. A number of outward signs, such as icteric and bronze colour of the skin, exophthalmos, pulsation of the carotid arteries and jugular veins, etc., may aid diagnosis. Besides vision, other organs of sense are used for examination; for example, the sense of smell may help detect the onset of uraemia (odour of ammonia), diabetic coma (odour of anise apples), or pulmonary gangrene (foetor), etc.

An examination should be carried out according to plan and to a number of established rules. One should use diffuse daylight or daylight bulbs, preferably from a side, because this will throw the outlines of various bodily parts into greater relief, better reveal pulsation of vessels, various rashes, etc. The first step is to determine the position of the patient, his general condition, the state of the outer covering. This is followed by examination of individual parts of the body—the face, head, neck, trunk, extremities, etc.

Position of the patient. The patient's position may be active, forced, and passive. In an active position the patient is able to sit up without outward help, turn on his side, etc. In a forced position the patient assumes the posture bringing him utmost relief. For example, cardiac patients with a severe circulatory disorder constantly maintain a sitting or half-sitting posture. Patients with exudative pleurisy lie on the affected side; those with an attack of cholelithiasis or nephrolithiasis are shifting frequently in search of an easier position; and patients suffering from an attack of pain due to gastric ulcer assume what may be called a shoe-maker's posture or lie on their stomach. In a passive position the patient lies motionless completely unable to change his position without outward help. These are patients enfeebled and wasted by an emaciating disease, as well as those in a state of unconsciousness. It should be kept in mind that sometimes even a severely ill person may be feverishly active when excited. Some persons over-anxious about their health are prone to take to their beds whenever they feel a little indisposed, while a person careless of his health may be up and about for a long time even when seriously ill.

The state of mind. A patient may be in a clear or confused state of mind, in stupor (mental apathy), sopor (mental sleep), or coma (total unconsciousness).

A patient with confused consciousness has a cloudy, stunned mind, gives delayed and slow, though sensible, answers, and sometimes is unable to give any answer at all. In stupor the patient is overwhelmed by mental apathy from which he can be roused briefly by a loud call or shaking, but his speech will, nevertheless, be senseless.

Extremely intense stupor is known as sopor, when the patient is half asleep, as it were. He is indifferent to his surroundings, unable to answer questions, but his reflexes are retained. The comatose condition is characterized by total unconsciousness, lethargy, muscle relaxation, and loss of sensitivity and reflexes.

A disorder of consciousness may be brief or lasting. In the former case it follows a temporary disturbance in the cerebral circulation and is known as syncope, i.e., fainting; in the latter case it is consequent upon an affection of the central nervous system, cerebral haemorrhage, toxaemia (in infectious diseases), etc. Several infectious diseases, such as lobar pneumonia or typhus are sometimes attended with a severe disturbance of consciousness manifested by raving (delirium) and acoustic and optic hallucinations, when the patient attempts to jump out of bed and run away, talks to an imaginary opponent, etc. Such delirium may be quiet or violent.

Facial expression. In patients with fever the face has a peculiar excited expression—facies febrilis. The cheeks are red and the eyes shining. In emaciating hectic fever (febris hectica) the sunken cheeks show red spots, the eyes are wide open and moist. Lung tuberculosis lends a hectic flush to the cheeks, while in lobar pneumonia the cheek turns red on the affected side. In a number of diseases accompanied by a severe metabolic disorder as well as in acute peritonitis and severe diarrhoea emaciating and dehydrating the body, the face acquires a characteristic expression (facies Hippocratica); the eyes are sunken, the nose is sharp, the skin is pale, the lips are cyanotic, and the face is covered with drops of cold perspiration. The face may look like a mask, i.e., become stiff, motionless, which occurs in a decreased thyroid function as well as in encephalitis. The face with a permanent grimace resembling a laugh is found with tetanus, and is called **risus sardonicus—sardonic grin.**

The eyes. The eyes should be inspected thoroughly. In disturbance of fat metabolism flat yellow patches known as xanthomas appear on the eyelids. Protrusion of the eyeball (exophthalmos) is one of the signs of Basedow's disease (exophthalmic goitre). Oedematous eyelids are encountered with generalized oedema in persons with a kidney or heart disease. Constriction of the pupils is found with uraemia and morphine and alcoholic intoxication; dilation, in atropine poisoning. The size and identical colour of the retinas and their reaction to light are also of importance.

Inspection of the integuments. General examination of the patient is followed by inspection of the skin and mucous membranes. Normally the skin colour depends on the degree in which the blood is visible through the superficial layers of the skin. A change in the skin colour may be due to the following three principal factors: (a) the colour of the blood; (b) the patency of the blood vessels; (c) the thickness and transparency of the skin. The skin colour may change when the skin is thickened, hardened and has lost its transparency, when there is a decrease or increase in the level of haemoglobin or erythrocytes, constriction or dilation of vessels. In certain cases, however, a change in the colour of the skin may be due to the deposition of colouring matter (bilirubin, etc.) within its depth.

Pallor. Pallor of the skin with a normal composition of the blood may be temporary or permanent. Temporary pallor occurs with a spasm of the skin vessels caused by excitement, cooling, chilling, etc. In acute anaemia resulting from internal haemorrhage pallor is rather quick to appear. A sudden onset of pallor is also encountered in case of a sharp decrease in the amount of blood in the skin vessels and blood congestion in the vessels of the abdominal cavity, which takes place in fainting, collapse, etc.

Persistent pallor is more frequently seen with anaemia. The skin colour acquires variable hues: that of alabaster in chlorosis, a sallow hue in cancer, the hue of "coffee with milk" in subacute bacterial endocarditis, a brownish-yellow hue in pernicious anaemia, etc. Persistent pallor is also seen with a prolonged spasm of the skin vessels.

Temporary redness of the skin is encountered after alcoholic and atropine intoxication, in fever, after taking a hot bath, administration of vasodilating drugs, and in strong emotions. Excitement often causes a flush on the face, neck and upper part of the chest, especially in women.

Persistent redness of the skin is seen with an excessive increase in the number of erythrocytes, but it may also occur with a normal composition of the blood. The latter takes place in patients with hypertensive disease, alcoholics and persons protractedly and constantly exposed to cold, wind and light, because in such cases the vessel walls lose their elasticity.

Cyanosis. Cyanosis of the skin may be generalized and local. The former is due to inadequate arterialization of the blood, i.e., oxygen insufficiency and excess of carbon dioxide. Generalized cyanosis is encountered with diseases of the heart, when the latter's inadequate function results in a slow-down of the blood current. In such cases the tissues intensively absorb oxygen from the blood and saturate it with carbon dioxide just as rapidly. This accounts for the bluish colour of the tip of the nose, earlobes, fingers and toes in cardiac patients.

Cyanosis of the face and extremities and sometimes of the entire skin surface may be seen with a large number of lung diseases. The destruction of lung capillaries deranges gas exchange with the air, the blood is insufficiently oxygenated and its carbon dioxide content and colour are very close to those of venous blood. Generalized cyanosis is sometimes encountered with certain congenital heart defects, particularly stenosis of the pulmonary artery, chemical poisoning, as with potassium chlorate, aniline, nitrobenzene, as well as in hypersensitivity to certain medicinal substances (sulpha drugs) when haemoglobin converts into methaemoglobin.

Local cyanosis of the skin follows cessation of the venous drainage from a given region in case of obstruction of a vein.

Jaundice (icterus). Yellow pigmentation of the skin is due to an excess of bile pigments in the blood and their deposition in the skin. The amount of bilirubin in the blood increases when the drainage of bile from the liver into the duodenum is hampered. This difficulty is due to several causes: obstruction of the common bile duct with bile calculi and newgrowths; compression of the duct by a malignant tumour of the head of the pancreas or Vater's ampula; the presence of adhesions and inflammatory changes in the small biliary ducts. In such cases bile is retained in the biliary ducts and in the liver and only part of it enters the blood. This form of jaundice is known as mechanical or obstructive. In diseases of the liver cells the bile often enters not only the biliary canaliculi but also the blood vessels. This form of the disease caused by an affection of liver parenchyma is called parenchymatous jaundice. There is also haemolytic jaundice which is due to an intensified disintegration of erythrocytes and profuse destruction of haemoglobin from which bilirubin forms in the body. The latter form of jaundice is encountered with congenital and acquired instability of erythrocytes, potassium chlorate, chloroform, and phenol poisoning, severe infections, malaria, etc. The skin turns yellow also after taking picric acid per os as well as during acrichine (quinacrine) and tripaflavine (acriflavine) treatment. In such cases the amount of bilirubin in the blood is normal, the urine contains no bile pigments, and the colour of the palatal mucosa, the sclera and the inner covering of the lower lip is unchanged, whereas in genuine jaundice they take on a yellow colour of variable intensity.

Pigmentation of the skin. Pigmentation or dark colouring of the skin is encountered both in healthy and diseased subjects. Intense pigmentation occurs in exposure to sunrays (suntan) and in unclean persons, especially in places of the body subjected to friction. after the application of hot-water bottles, scratching, itching, etc. A bronze colour is observed in affection of the adrenals, a black colour in malaria running a protracted course, when melanin is deposited in the skin (melanosis). A dark colour of the skin with a leaden-grey hue is seen in patients who have undergone prolonged treatment with preparations of silver, which lend a characteristic colour to the skin (argyrosis).

Sometimes, circumscribed patches of local pigmentation appear on the skin, such as freckles (ephelides) and birth-marks (naevi). Diseases of the female genitalia may be attended with chloasma (chloasma uterinum), while pregnancy, with an intesified pigmentation of the nipples and the median line of the abdomen as well as of the cheeks and sides of the forehead (chloasma gravidarum). Complete absence of normal pigment in the skin is called albinism (albus—white), loss of pigment in isolated parts of the skin is known as vitiligo. Tiny white spots, parts of the skin devoid of pigment and appearing after skin rashes, are called leucoderma.

During inspection attention should also be paid to the presence and character of scars, if any. Scars of a stellate shape indrawn and adherent to the underlying tissues are left by syphilitic lesions, round pock-marks on the face are traces of smallpox. Scars on the sides of the abdomen are left by pregnancy and pituitary tumours (Itsenko-Cushing's disease); similar scars are encountered after obesity and subsidence of large oedemas. Surgical operations also leave scars at the sites of incision; these scars have a regular form and are usually linear or arch-shaped.

Haemorrhages in the skin. Skin haemorrhages are encountered in patients with skin, infectious and internal diseases, traumas, etc. Haemorrhage in the skin, the so-called bruise, occurs in healthy persons as a result of a blow, compression, or bite as well as in persons with predisposition to bleeding, in the latter case without any apparent cause. A bruise, which at first has a purple colour, after a time turns green-yellowish and soon disappears without leaving a trace. Haemorrhages in the skin which fail to disappear upon pressure may be of a variable size and form, now seen as punctate petechiae, now as large patches and bands. They often attend septic diseases, acute anaemia, jaundice, scurvy, diseases of the blood with a quantitative and qualitative thrombocyte deficit, etc. A large-patched redness of the skin known as erythema occurs with rheumatic fever, erysipelas, and urticaria (nettle-rash). In erysipelas erythema spreads very quickly, and is markedly circumscribed. In nettle-rash the skin is covered with itching red spots and white blebs rising above its surface, containing serous exudate and resembling blisters caused by nettle stings. Nettle-rash occurs in patients with intolerance of some foodstuffs, serum disease, food poisoning, after blood transfusion, etc. Eruption of red spots on the skin is also encountered in persons hypersensitive to certain medicinal substances (for example, iodine, bromine), or exposed to their toxic effects (sulpha drugs, penicillin).

A redness of the skin appearing as a circumscribed inflammatory dilation of the capillaries is known as roseola. This is a round pink or bright-red spot 2-3 mm in diameter. It disappears upon pressure and reappears after pressure is stopped. The presence of roseolas is of great diagnostic importance. They erupt in a small number in typhoid fever and paratyphoids on the skin of the abdomen and the lower part of the thorax on the 7th-10th day of the disease and disappear without leaving a trace in 3-6 days' time. In typhus, roseolas erupt on the 4th-5th day of the disease. They are more numerous and smaller than in the case of typhoid fever and cover the skin of the trunk and extremities. They disappear in 5-6 days or burst in their centres to leave petechiae, which persist during 12-16 days.

Peeling of the skin. Certain rashes which usually do not extend to the deep layers of the skin cause desquamation, i.e., peeling of the skin, the character of which is of great diagnostic value. For example, measles is characterized by branny desquamation, scarlet fever by the shedding of large scales, etc. Peeling of the skin is also encountered in tuberculosis, diabetes mellitus, emaciation, senility and poor care of the skin.

Bedsores. The skin may also be covered with erosions resulting from discontinuity of its superficial layers (in trauma, rupture of vesicles), and with ulcerations due to disturbances extending to the deeper layers of the skin. Deep ulcers developing in bed-ridden patients in the region of the sacrum, the buttocks and in places subjected to prolonged pressure are called bedsores (decubitus). Bedsores may not only affect the skin and subcutaneous adipose tissue but also erode the muscles. They are produced by prolonged pressure against these parts, which causes their exsanguination and mechanical irritation.

The hair and nails. During inspection attention should also be paid to the condition of the hair and nails. With suppurative processes in the bronchi and lungs the nails may become curved over the finger-ends. Excessive brittleness and loss of hair occur in myxoedema, diseases of the pituitary body and as a sequel to many infectious diseases. Hairs come out in ringworm (favus) and seborrhoea. Loss of hair on the scalp (alopecia areata) and brows, leaving isolated bald patches, occurs in syphilis.

The nails may become flat, frail and lustreless, with spoon-shaped depressions, or curvature over the finger-ends, and undergo other changes as a result of trophic disorders in certain diseases of the nervous system, the skin and the internal organs.

PALPATION OF THE INTEGUMENTS AND MUSCULO-SKELETAL SYSTEM

Palpation may give valuable information on the dampness and elasticity of the skin and the state of the subcutaneous cellular tissue.

Dampness of the skin. The degree of dampness of the skin depends on perspiration. Increased dampness is noted in many febrile diseases (rheumatism, pulmonary tuberculosis, etc.), in a sudden drop of body temperature (lobar pneumonia, relapsing fever), in thyrotoxicosis, and in severe functional insufficiency of the cardiovascular system. In the latter case, the perspiration is cold and sticky, while the skin turns pale, whereas in fever perspiration appears on the reddened skin.

Local increased perspiration on a half of the scalp, hands, and legs occurs with certain diseases of the nervous system. Dryness of the skin is caused by an excessive loss of fluid by the body, prolonged wasting diarrhoea, diabetes mellitus and diabetes insipidus, uraemia or by disturbances in the absorption of fluid during vomiting and constriction of the pylorus.

Skin turgor. The turgor or elasticity of the skin depends on the fullness of the blood vessels, and content of fluid and lymph in the skin. Decreased elasticity is seen after prolonged loss of fluid during a flux as well as in emaciating diseases. In old age the face becomes wrinkled as a result of dehydration of the tissues. Folds on the skin with a decreased elasticity fail to smooth out for a long time.

Subcutaneous cellular tissue. Palpation may help determine the amount of fat in the subcutaneous cellular tissue.

The skin with a thick layer of fat resists folding, whereas that with a thin fatty layer can be folded easily. Attention should be paid to the character of distribution of fat. In affections of the pituitary body fatty tissue is deposited mainly on the buttocks and thighs, in ovarian adiposity, on the abdomen and around the mammary glands. Palpation may also reveal fatty tumours (lipomas) in the subcutaneous cellular tissue.

Oedema. Oedema of the subcutaneous cellular tissue of the entire body is known as anasarca. It is encountered in diseases of the heart and kidneys and in cachexia. Renal oedema appears at first on the face and eyelids and later spreads throughout the body.

Cardiac oedema primarily appears in the lowest parts of the body, located far from the heart. Cachectic oedema (in cancer, alimentary dystrophy) spreads throughout the body. A large oedema is visible to the naked eye, a slight one is recognized by pressing the skin against the underlying bone with the finger which leaves a depression if oedema is present. Slight oedema is known as pitting oedema.

Oedema may be fluctuating or rigid and hard. There may be oedema of angioneurotic origin which occurs as a result of a sudden local dilation of vessels. It is mainly confined to the face and extremities. Unilateral oedema on a leg is consequent upon a disturbed drainage of venous blood and lymph (in thrombophlebitis, after fractures of the shin bones, erysipelas, etc.). Inflamed joints are surrounded by oedematous subcutaneous cellular tissue. The skin in this region is red, lustrous and hot to the touch.

Palpation of the muscles. Palpation of the muscles may give an idea of the patient's muscular development. In emaciating diseases, the muscles undergo atrophy, i.e., they become thin, flaccid, and their contractile ability is considerably reduced. Diseases of the joints, bones and peripheral nerves are attended with limited atrophy of the muscles in the affected region in consequence of trophic disorders. Complete muscle paralysis may also be of cerebral origin, for example in cerebral haemorrhages, traumas and tumours of the brain. Unilateral paralysis of the upper and lower extremities is called hemiplegia, a weakening of the motor function of an extremity on one side of the body is known as hemiparesis.

Palpation of the lymph nodes. Examination of the lymph nodes serves to determine their size, consistency, mobility, and whether they are fixed to the skin and underlying tissues. Lymph nodes may be felt on the front and sides of the neck, in the submaxillary and axillary fossae, in the inguen, the popliteal fossa, and in the antecubital fossa (characteristic of syphilis). Lymph nodes become enlarged in the presence of various inflammatory processes in the region from which lymph is supplied to them. For example, in dental diseases the submaxillary lymph nodes become enlarged in case of an infected wound of the shank or thigh—in the inguinal lymph nodes, etc. Enlargement of the lymph nodes is often consequent upon a metastatic spreading of carcinoma. In particular, in stomach cancer an enlarged lymph node is sometimes found between the two heads of the sternocleidomastoid muscle. Enlargement of the lymph nodes is often seen with systemic diseases, such as lymphogranulomatosis, leukaemia, tuberculosis. In tuberculosis of the lymphatic glands the latter become adherent to one another and to the underlying tissues and to the skin. In caseous decay they suppurate and form fistulas, i.e., channels through which pus is discharged outside. In such cases the pus is sometimes found to contain tuberculous mycobacteria.

Palpation of the bones and joints. Palpation of the bones may reveal their tenderness, as well as the presence of deformities, clubbed fingers and toes, fractures, bone calluses and tumours. Various malignant newgrowths in the internal organs (cancer, sarcoma, hypernephroma, myeloma, etc.) often produce metastases to the bones, which cause intense pain and sometimes result in bone fractures. In examination of the joints attention should be directed to their form, mobility, tenderness, presence of exudate in the joint cavity, and colour of the overlying skin.

MEASUREMENT OF THE BODY TEMPERATURE AND TYPES OF FEVER

The temperature of the human body depends on generation of heat as a result of assimilation of ingested food, decomposition of cells of the body itself, and the degree of heat irradiation into the atmospheric air. Normally body temperature ranges from 36 to 37°C, is lower in the morning and higher between 5 and 9 p.m. As a rule, the temperature in a healthy person slightly rises after physical exertion, a hearty meal, especially of meat, in an overheated room, and after excitement. It drops somewhat during sleep and rest. A change in the temperature may indicate a morbid condition of the body. The temperature is considered low (hypothermy) when it is under 36°C. Hypothermy may be due to emaciating diseases or chronic intoxications. It is encountered in persons with heart diseases in the stage of a circulatory disorder, after a considerable blood loss, as well as during recovery from acute infectious diseases. An increased temperature (hyperthermy) occurs in febrile diseases, haemorrhages, comminution of tissue, bone fractures, and thyroid and adrenal hyperfunction. An increase in the body temperature affects the general condition of the patient who feels jaded, with dryness and an unpleasant sensation in the mouth. The urinary discharge markedly lessens, and an intense thirst is noted sometimes. The appetite is reduced, metabolism intensified, and the patient loses flesh. A temperature increase of 1° causes acceleration of the pulse rate by approximately eight beats per minute, except for certain diseases, such as

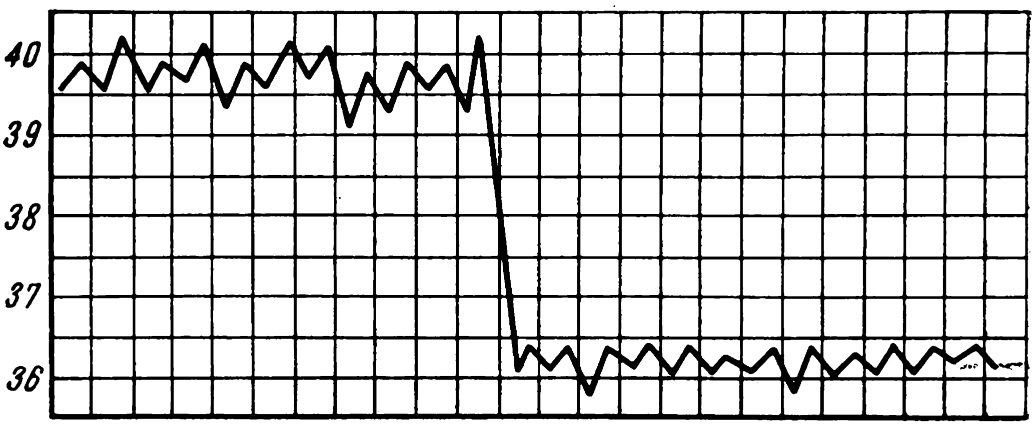


Fig. 4. Continued fever with a critical drop of temperature

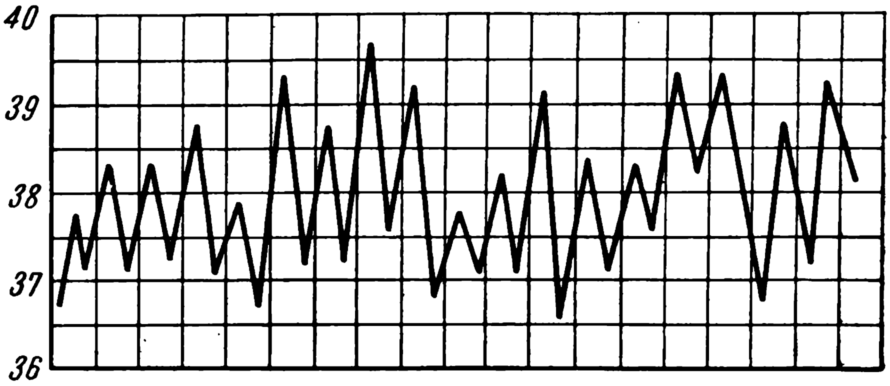


Fig. 5. Remittent fever

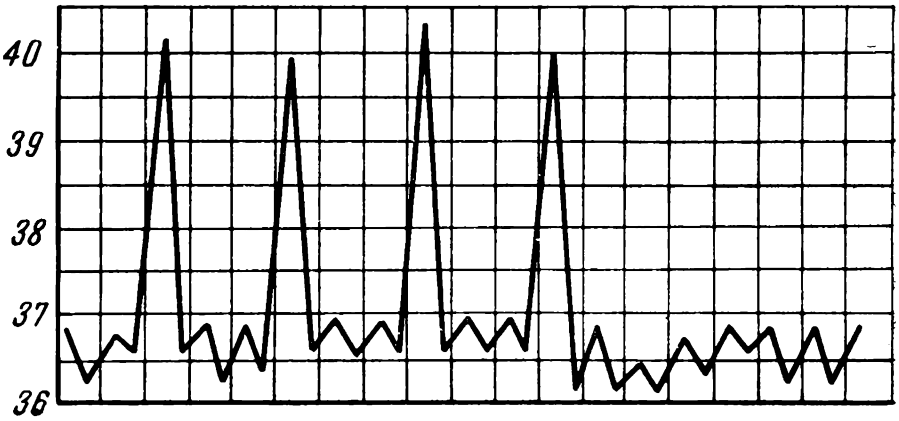


Fig. 6. Intermittent fever

meningitis and enteric fever, in which the pulse rate lags behind the temperature rise. The respiration also becomes more frequent: at a temperature increase of 1° the respiratory rate increases by four movements per minute. A sudden rise of temperature is accompanied by severe chills which last from 30 minutes to 1 hour and sometimes longer. The patient shivers with cold and his teeth clatter. These sensations are caused by a sudden constriction of the skin vessels as a result of which the skin turns pale and the nails bluish, and a goose flesh skin occurs. During a gradual increase of temperature the above symptoms are either absent or are unmarked, but the skin is flushed and the patient fells hot. A sudden drop of temperature to normal or below normal is accompanied by intense perspiration and considerable debility.

Fever has the following gradations: temperature ranging from 37 to 38°C is known as subfebrile; from 38 to 39°C as mildly febrile; up to 40°C, febrile; and over 40°C high-grade.

The type of fever depends on the daily variations of body temperature during a disease. Certain diseases are characterized by a specific temperature curve. There are eight types of fever, which are as follows:

1. *Continued fever* (febris continua) in which the difference between the temperature in the morning and in the evening does not exceed 1°C. It is seen with lobar pneumonia, typhus, etc. (Fig. 4).

2. *Remittent fever* (febris remittens) in which daily temperature variations are equal to 1-2°C and more. The morning temperature is above 37°C. It is found in purulent diseases, and often in pulmonary tuberculosis (Fig. 5).

3. *Intermittent fever* (febris intermittens) in which the temperature rises to 39-40°C and higher and usually drops to normal or subnormal within a few hours. It recurs every other day or with two or three intervening normal days and is characteristic of malaria (Fig. 6).

4. *Hectic fever* (febris hectica) in which temperature fluctuations are much larger than in remittent fever, reaching 2-4°C. It is marked by a quick drop of temperature to or below normal, and is accompanied by profuse, emaciating perspiration. It is found in severe forms of pulmonary tuberculosis and sepsis (Fig. 7).

5. *Recurrent fever* (febris recurrens) is characterized by a sudden rise of temperature to a high level which in a few days' time is followed by a temperature drop to normalcy lasting for several days and by an onset of another period of fever with a subsequent temperature decline. Such attacks of recurrent fever may occur up to five times. They are characteristic of relapsing fever (Fig. 8).

6. *Inverted fever* (febris inversa) or the inversive type of fever (typus inversus) in which the temperature in the morning is higher than in the evening. It is found with pulmonary tuberculosis, sepsis and brucellosis.

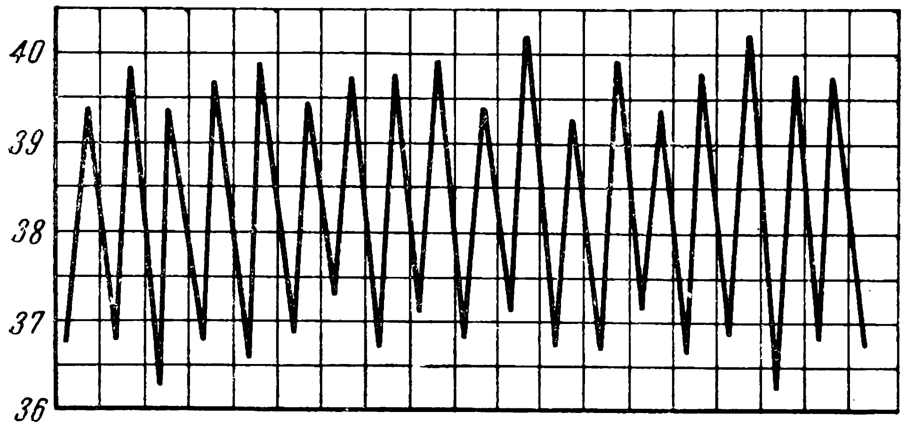


Fig. 7. Hectic fever

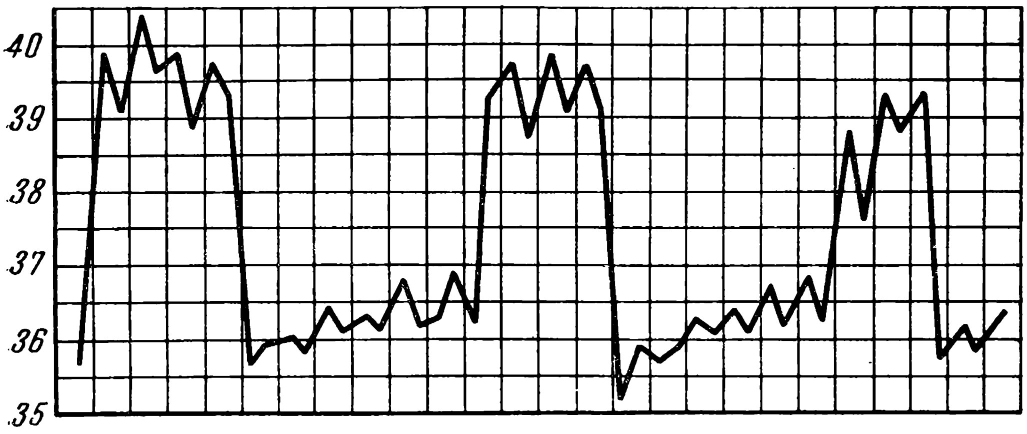


Fig. 8. Recurrent fever

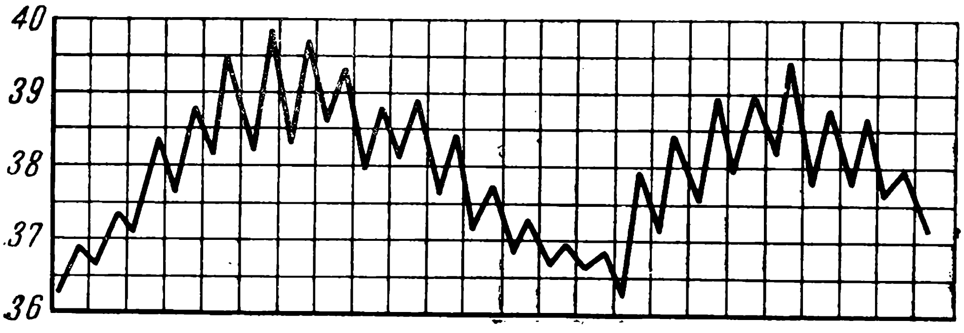


Fig. 9. Undulant fever

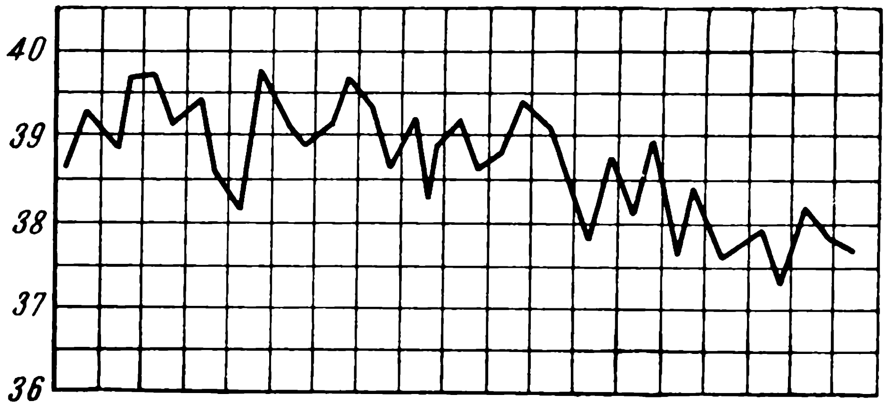


Fig. 10. Irregular fever

7. *Undulant fever* (febris undulans) which is characterized by a gradual increase of body temperature followed by a gradual decline to normalcy. The feverless period is succeeded by a gradual rise of temperature and by another drop to the normal level.

It differs from recurrent fever for its gradual increase and decline of temperature, and is found in brucellosis (Fig. 9).

8. *Irregular fever* (febris irregularis) which is the most frequent. It is characterized by irregular diurnal variations of different duration, and occurs in rheumatic fever, dysentery, influenza, sepsis, rheumocarditis, etc. (Fig. 10).

The period of fever is known to have three stages: (a) the stage of temperature increase (stadium incrementi) in which the temperature rises suddenly or gradually within several hours or days, depending on the character of the disease; (b) the stage of high-grade fever (stadium fastigium) in which fever lasts from several hours to several days and weeks, depending on the character of the disease; (c) the stage of temperature decline (stadium decrementi).

The character of temperature decrease may also vary. In some cases, the temperature falls to normal and even subnormal figures within 12-24 hours. This type of temperature fall is known as critical or crisis. In other cases the temperature declines to normal within several days. This type of abatement of fever is known as lytical or lysis. Sometimes, a crisis is preceded by a brief rise of temperature (perturbatio critica). Before the lytical decline the diurnal temperature variations often exceed 1° for several days. This is the so-called amphibolic stage. The critical decline of temperature is characteristic of lobar pneumonia, malaria, typhus; lytical decline—of bronchopneumonia and enteric fever; the amphibolic stage is characteristic of enteric fever.

The technique of body temperature measurement. The body temperature should be measured at least twice daily; in the morning between 7 and 8 a.m. and in the evening between 5 and 6 p.m. The temperature is measured by means of a maximum Centigrade thermometer with graduations from 35 to 42°. A maximum thermometer is one in which the mercury remains at the maximum height reached during a measurement, and is brought down only by vigorous shaking. After each measurement a thermometer should be shaken and disinfected. The temperature in adults is usually measured by inserting a thermometer under the armpit. Before this procedure the armpit should be wiped dry, because the skin moisture may reduce the thermometer readings. The thermometer is allowed to remain under the armpit for ten minutes. Sometimes the temperature has to be measured in the rectum, keeping in mind, however, that the rectal temperature is 1° higher than that under the armpit.

In addition to a general change, a local change of temperature due to a circulatory disturbance in certain parts of the body is noted sometimes. A reduction of temperature in both extremities is found with disturbance of peripheral blood circulation in cardiovascular insufficiency. An increase of the temperature in one extremity occurs in inflammation of an artery (endarteritis); an increased skin temperature around the joints is most frequently noted during inflammatory processes within the latter.

HABITUS

The patient's general physical appearance and body-build are known by the term habitus. The characteristic features of the body-build and general appearance may help to draw a conclusion on the state of health.

Straight gait indicates good health and training. Atactic (incorrect) gait is seen in tabes dorsalis (locomotor ataxia), which is a syphilitic affection of the spinal cord. In this disease the patient lifts his leg with difficulty, and making a wide circling movement sets it down on the heel. After brain haemorrhage the patient drags his leg, lesions of the cerebellum cause unsteady gait, etc.

Inspection of the patient also serves to determine his type of constitution. Constitution is the sum total of congenital and acquired morphologic and physiologic characters of the human being. It is subject to change under the influence of various environmental factors.

Among the various classifications of the types of constitution the best one is that drawn up by M. Chernorutsky who distinguishes the following three types: asthenic, hypersthenic, and normosthenic.

The *asthenic* type (*sthenia*—strength, vigour) is characterized by long and thin extremities, narrow palms and thin fingers, and a thin and slender skeleton. The neck is long and thin, the shoulders are narrow, the chest is long and narrow, with wide intercostal spaces. The shoulder-blades often jut out from the thoracic case, the epigastric angle is acute, the abdomen is small, the muscles are underdeveloped, and the skin is thin and pale. The subcutaneous cellular tissue is underdeveloped, the diaphragm position is low, and splanchnoptosis is frequent. In persons of the asthenic type metabolism is intensified, and the arterial pressure diminished. The principal characteristics of their body-build are considerably increased vertical proportions in respect to the horizontal ones. They often have a floating tenth rib (costa decima fluctuans).

The *hypersthenic* type is the exact opposite of the asthenic type. The vertical proportions are diminished with regard to the horizontal ones. The extremities are short and thick, the head is large and round, the face is broad, the forehead is high, the neck is short and thick. The chest is wide and short, the shoulders are broad and straight, the ribs lie horizontally, the intercostal spaces are narrow, the epigastric angle is obtuse, the abdomen is enlarged. The bones are wide and heavy, the diaphragm position is high, the stomach lies in a high transverse position, the intestine is long, metabolism is decreased, and there is a trend to an increase in the arterial pressure.

The *normosthenic* type, which is the most frequent, occupies an intermediate position between the two latter types. It is characterized by a correct correlation between the vertical and horizontal proportions of the body. There is a variety of deviations from the above-described types.

According to Ivan Pavlov's theories, the basis of the constitution is the nervous system, since it ensures the integrity of the body and its communication with the external environment. Pavlov subdivided the higher nervous activity in animals into the following four principal types: (1) strong, unrestrained; (2) strong, steady, slow; (3) strong, steady, quick; (4) weak.

When laying down these four types, Ivan Pavlov was guided by four types of temperament: the choleric (bilous), the sanguine, the phlegmatic (lymphatic), and the melancholic, which had been described by ancient Greek physicians, but he put this classification on a physiologic basis. He also distinguished specific types characteristic of a particular person: the artistic, the intellectual, and the average types. According to Pavlov's theory, the artistic type is distinguished by prevalence of the first signal system over the second one. A person of this type thinks mainly in images and has concrete thinking. Persons of the intellectual type are the direct opposite of the former and think in the abstract. A person of the average type has both the first and the second signal systems equally developed, and therefore his thinking is equally based on concrete images and abstract notions.

Ivan Pavlov considered the type of higher nervous activity the principal part and the basis of man's general constitution.

ANTHROPOMETRIC EXAMINATION

The general structure of the body is determined by means of anthropometry (Gr. *anthropos*—man; *metron*—measure), i.e., measurement of a person's height, thorax circumference, and body weight.

The stature. People may be tall, short or of medium height. A stature of not more than 100 cm is considered dwarfish, and that above 190 cm gigantic. A person's height is measured by means of an upright measuring rod, graduated in centimetres, and provided with a sliding horizontal lath. The patient should stand erect, his heels, buttocks and shoulder-blades touching the measuring rod, the upper margin of the external auditory meatus and the outer angle of the eye (outer canthus, palpebral angle) lying in one horizontal line. The height is determined from the indication of the horizontal lath at the point of contact with the top of the head.

The thorax. The thorax circumference is measured at the moment of exhalation by means of a tapeline graduated in centimetres, the patient having his arms lowered and breathing calmly. The tapeline is placed so that it passes just beneath the shoulder-blade angle at the back and along the sixth rib at the front. For practical purposes very important is the relation of body length to thorax circumference, the latter normally being equal to a half of body length. The diameter of the thorax should also be measured.

There are the anteroposterior and the transverse diameters which are determined with a pair of compasses by measuring the sterno-vertebral, or anteroposterior, and the transverse, or costal, dimensions.

The weight. A person may be normally nourished, obese, lean or cachectic. The degree of nourishment depends on the development of the subcutaneous fat and cellular tissue. Normally the layer of this tissue on the abdomen is not more than 1 cm thick. In undernourished persons the subcutaneous fat and cellular tissue is quite underdeveloped, the abdomen is hollow, the intercostal spaces indrawn, with deep supra- and infraclavicular fossae. The utmost degree of emaciation is known as cachexia. Excess fat in the subcutaneous cellular tissue is responsible for thick folds on the abdomen, neck, and chest, and for the disappearance of depressions on the chest. In obesity one should take note of its character, i.e., uniform or irregular deposition of fat on the abdomen, buttocks, and other parts of the body.

Normal body weight is equal to the number of kilograms derived by deduction of 100 from the figure indicating a patient's height. For example, the normal weight of a person standing 175 cm is 75 kg. An insignificant deviation in both directions is not yet an indication of obesity or undernourishment.

Body weight is measured upon admission of patients to a medical institution and before discharge as well as (insofar as practicable) weekly, and is an indispensable procedure in clinical examination. Patients are weighed in the morning undressed and on an empty stomach, after evacuation of the urinary bladder and, whenever possible, of the bowels. The weight measurements are recorded in the case history. Regular records of body weight may be an important guide in following the course of a disease. It should be borne in mind that considerable variations in body weight are most commonly seen with accumulation of fluid in the body or with intensive resolution of oedema, which occur, in particular, in heart and kidney diseases.

X-RAY EXAMINATION

The x-ray room is used for roentgenoscopy, roentgenography and x-ray therapy. Its standard equipment is an x-ray unit consisting of a vacuum tube, tube supports, a place for the patient, a filament transformer, and a high-voltage transformer. The bulb-shaped x-ray tube made of glass contains two sealed cathode and anode electrodes. The cathode electrode has a tungsten spiral heated to 2,500°C by means of the filament transformer. This results in the generation of negative-charge electrons, which flow at a great speed to the anode electrode owing to the high voltage of the electric field. These electrons are instantly brought to a standstill on the anode, and their tremendous kinetic energy turns into x-rays. The x-ray unit is provided with a shield to protect the examiner against x-rays.

X-rays, discovered by Roentgen in 1895, have the following three basic properties: (a) they penetrate through solid bodies by which they are partly absorbed; (b) they cause fluorescence of certain substances; (c) they decompose silver bromide on photographic films and plates.

These properties make possible roentgenoscopy, roentgenography and x-ray therapy of certain diseases. For roentgenoscopy the parts of the body subject to examination are placed between the x-ray source and the screen. The latter is made of cardboard coated with a fluorescent substance. In the Soviet Union, zinc-cadmium-sulphide-coated screens are used.

The tissues and organs of the human body have a variable consistency and differ for their capacity to absorb x-rays. The largest amount of radiation is absorbed by solid bodies, while soft tissues and air are the least capable of such absorption. That is why bones subjected to x-raying throw an intensely dark shadow on to the screen. A dense shadow is produced by a vascular bundle and the liver; a less intense shadow, by the stomach. The air absorbs an insignificant quantity of x-rays. Therefore, roentgenoscopy of organs containing air shows pale areas which are the lighter the more air is contained in an organ.

Fluids also absorb x-rays very intensively, so the intensity of a shadow cast by a fluid is directly proportionate to its amount.

The body contains a considerable number of adjoining organs of identical consistency. In conventional roentgenoscopy their shadows merge, which makes x-ray examination useless. Even in such cases, however, a contrast chemical substance harmless to health may be introduced into the organ under examination to produce a sharp distinction between the latter and the surrounding organs and tissues.

Another method is injection of air or oxygen into cavitary organs, such as the stomach, intestine and abdominal cavity because air and oxygen produce very pale areas on the x-ray screen, thereby forming a sharp contrast between an air-filled organ and the adjoining parts.

Bodily organs are often arranged in layers at different levels or closely adjoin one another. In such cases, the x-ray screen shows a shadow produced by the passage of x-rays through all organs. This shadow is the sum total of all shadows cast by organs adjoining each other. For example, in an x-ray image of the lungs, the lower parts are paler than the apices, although in either case it is air that is responsible for the paleness. The lung apices are less pale not only because they contain less air but also because of the overlying thick muscular shoulder girdle.

To prevent summation of shadows, x-raying is performed in an oblique or lateral direction, since this will separate shadows, which are otherwise cast one upon another when seen in a frontal projection. X-raying in oblique directions makes it possible to determine the size of an area as well as pathologic changes therein. This is based on the fact that the size of an organ as shown on the x-ray screen is the larger the greater the distance between the latter and the organ under examination. X-raying in different projections is also employed to locate the organs examined.

Fluorography is a method of examination widely used to detect pulmonary tuberculosis running a masked course. It essentially consists in photography of the x-ray screen image of the lungs followed by reading of the films on a filmoscope. About 100 films can be taken in one hour. If tuberculous lesions are discovered in the lungs conventional x-ray photography is undertaken.

Fluorography enables a mass-scale x-ray survey to be carried out within a brief space of time. Pictures obtained by means of fluorography are known as fluorograms.

During the past few years, x-ray kymography has been employed for the study of the functions of mobile organs, such as the heart and stomach, as well as of larger vessels, ribs and the diaphragm.

Roentgenography is a method by which pictures are taken on photographic film or plates coated with silver bromide. X-rays decompose silver bromide in the same way as visible radiation does. For x-ray photography of a certain part of the body the object to be examined is placed between the x-ray tube and photographic film or plate protected against the decomposing effect of light. X-rays reduced in intensity during their penetration through various parts of the object photographed (air, tissue, kidneys) will have a variable effect on silver bromide, so that the degree of the latter's decomposition on photographic materials will vary also. After photography a film or plate is developed and fixed. In an x-ray photograph, as distinct from an x-ray screen image, dense substances are of a light colour, while soft tissues and air are dark.

X-ray photography is used to obtain a more detailed picture of slight pathologic changes, which are likely to be overlooked in roentgenoscopy but are clearly seen in an x-ray image. X-ray photos taken at different times illustrate the changing course of a morbid process.

An x-ray tube emits scattered rays, as a result of which the x-ray image is larger than the actual size of an object under examination. To bring the image size closer to the natural proportions of an organ the latter should be placed nearer to the screen or the plate. This will also make the shadow outlines more distinct. More accurate data on the size of an organ are obtained by orthodiagraphy. The x-ray image is produced by the central, non-scattered x-ray beam. Special screens provided with devices for recording the contours of an organ are used for this purpose.

Examination of the thoracic organs. Normally, the x-ray image of the lungs shows pale areas crossed by ribs. The x-ray image of the normal lungs is paler along the periphery than at the lung root and darker at the apices due to the thin layer of air contained in them and to the muscles of the shoulder girdle. The lung apices grow lighter in coughing. In one drawing a deep breath the lower parts of the lungs become paler still as a result of an increase in air volume. In roentgenoscopy the bronchial cross-sections appear as light circles with a dark rim. X-ray examination of the lungs is based on observation of changes in image transparency, the presence of shadows in light areas or a still greater increase of image transparency. Because of an increased amount of air in the lungs the translucency of a lung image may be diffuse or circumscribed.

A diffuse translucency is seen with emphysema and pneumothorax, a circumscribed one, with bronchiectasis, caverns and cavities. Diffuse or circumscribed lessening of the lung image transparency or shadowing of light areas is encountered when air is displaced from the lungs by a newgrowth or fluid. Lung cavities filled with fluid are found with abscesses and tuberculous caverns communicating with the environmental air through a bronchus. They appear as a dark-coloured rimmed bowl, with a variable horizontal level depending on the amount of fluid, and as a translucency above the level corresponding to the air sac. Roentgenoscopy also reveals both the shadows of adhesions and calcareous deposits in various parts of the lungs and lymphatic glands of the thoracic cavity in consequence of pulmonary tuberculosis.

An x-ray examination of the respiratory organs is carried out also with the aid of bronchoscopy. The method of bronchoscopy is as follows. After a preliminary dicaine (tetracaine) anaesthesia of the pharynx, a contrast substance, iodolipol, is introduced through a catheter into the bronchus to be examined, which is followed by photography of the lungs. Bronchography serves to reveal bronchiectasis and newgrowths. In the former case, the contrast substance filling a cavity produces the latter's image on the film. A newgrowth obstructing a bronchus produces the image of a bronchus coming short of the lung tissue. This image is termed amputation of the bronchus. A contrast substance introduced for bronchography is resorbed or expectorated within 1 to 3 days.

X-ray tomography of the respiratory organs shows not only the foci of disease but also the depth of their location. Body section tomography enables an x-ray picture of a particular layer to be obtained, while all the other layers located around, in front of and behind it are blurred because of the scattered x-rays.

Examination of the heart and vessels. X-ray examination is used to determine the size of the heart, its shape and position inside the thorax. The action of the heart and every one of its parts is studied by means of x-ray kymography.

A cardiovascular shadow projected on a screen has an irregular egg-like form and occupies a median position. It is located obliquely above the diaphragm, two thirds of it lying in the left half and one third in the right half of the thorax.

X-ray examination of the heart and vessels is performed in the anteroposterior direction, in the first oblique (torso turned right by 45°) and in the second oblique (torso turned left by 45°) directions.

The right-hand contour produced by anteroposterior roentgenoscopy is an irregular arc with two convexities. The upper arc extends from the 1st to the 3rd rib and is formed by the vena cava superior and the ascending part of the aorta; the lower arc is formed by the right atrium and sometimes, when the heart is in a vertical position, also by the right ventricle and the vena cava inferior.

The left-hand contour of the heart consists of four arcs. The upper arc lies on a level with the first intercostal space and corresponds to the aortic arch; the second and third arcs correspond to the pulmonary artery and the left cardiac auricle; the fourth arc corresponds to the left ventricle.

Enlargement of the second and third arcs of the left contour forms the so-called mitral configuration of the heart with a flattened heart outline, which is very typical of a mitral defect. Aortic valve insufficiency causes an increase in the first and fourth arcs at left, while the angle between the vascular bundle and the heart is retained. The heart silhouette grows larger leftwards and downwards, acquiring an aortic configuration comparable to the silhouette of a sitting duck.

X-raying makes it possible to determine the shape of the heart apex also. In hypertrophy of the left ventricle the apex is rounded. In persons with an asthenic constitution and a lowered diaphragm the heart lies in a vertical position, the heart shadow has small contours and seems to be diminished in size. When the diaphragm position is high (flatulence, obesity, pregnancy, etc.) the heart takes a recumbent or a transverse position, and may be contiguous to the diaphragm.

X-ray examination may also reveal the so-called drop heart (pendulous heart, cardioptosis), which looks like an appendage hanging from a vascular bundle and slightly touching the diaphragm.

X-raying may help detect acquired and congenital dislocations of the heart. Exudative or adhesive pleurisy and pneumothorax are accompanied by acquired dextrocardia, i.e., transposition of the heart to the right side of the thorax. Sometimes, the right half of the trunk, in addition to the heart, contains the spleen, and the left half, the liver, a congenital anomaly in which the viscera are changed from the normal to the opposite side of the body (situs viscerum inversus).

The presence of effusion in the heart sac (pericardium) produces a triangular shadow of the heart. Adhesions formed by resorption of exudate and calcareous deposits on the heart sac are responsible for dark contours surrounding the heart image, a condition known as armoured heart. X-raying also reveals changes in the aorta, which, when affected by sclerosis, gives a larger and darker shadow. In case of an aneurysm the aorta is diffusely or partially dilated in its ascending or descending portion, or in its arch.

The difference between an aneurysmatic dilation and a tumour is the absence of pulsation within the latter.

During roentgenoscopy in both oblique positions a light area appears in front of and behind the heart shadow. In the first oblique projection the light area lying between the shadow of the heart and vessels on one side and that of the backbone on the other side is known as the retrocardiac space. The light area in front of the heart is known as the retrosternal space. In the first oblique projection, changes in the images of the ascending aorta and the left atrium are seen more distinctly, enlargement of these structures leads to diminution of the retrocardial space. In the second oblique projection, changes in the aortic arch and its descending portion as well as enlargement of the right and left ventricles can be seen in relief.

Examination of the alimentary tract. X-ray examination of the digestive organs is carried out after the intestinal canal has been relieved of the faeces and gases, which largely interfere with the study of these organs. To avoid meteorism (gas distention of the abdomen), the patient is kept on a diet deficient in crude cellulose for 2-3 days before the examination. The intestine is cleansed twice with an enema. The first enema of 5-6 glassfuls of water is administered overnight, and a second one, 2-3 hours before the examination. The second enema is meant to remove the gases collected during the night and remnants of the faeces. One is not advised to use purgatives for cleansing the intestine lest they cause meteorism, intensify intestinal peristalsis, produce copious transudation into the intestines, and sometimes dislocate internal organs, which hinders detection of changes in the mucosa. Purgatives should be administered only for a stubborn constipation, when enema, and even siphonage, have proved inadequate. Outpatients may be given purgatives the day before an examination, if the facilities required for the application of a cleansing enema are unavailable.

Today, roentgenoscopy of the gastrointestinal tract is made by aid of a contrast substance, a suspension of barium sulphate, which is dispensed in packets. For a single x-raying session a patient is given 100 g of barium stirred in 150 ml of boiled water.

The oesophagus. For examination of the oesophagus, a patient is given both dilute and thick barium suspensions. When the suspension is being swallowed the examiner studies the peristalsis and antiperistalsis of the oesophagus, as well as the character of organic lesions, if any. X-ray examination helps detect foreign bodies in the oesophagus, such as fruit stones, fish bones, metal objects, etc.

A prolonged spasm of the oesophagus is accompanied by its considerable and uniform distention above the site of the spasm. The most common site of this distention is the space above the cardia. In an oesophageal spasm the contours of the narrowed portion are regular as distinct from those seen with a newgrowth, when the oesophageal walls are destroyed and corroded by the tumour. Carcinoma is usually located at the bifurcation of the trachea, at the cardia and above the aortic arch, i.e., in physiologically narrow places. Alkaline and acid burns of the oesophagus produce rough anatomical changes in the oesophageal wall and obstruction of the oesophagus, adhesion of barium causing a crumbly pattern of the wall. X-ray examination reveals congenital and acquired oesophageal diverticula.

The stomach. X-ray examination of the stomach is begun with a general survey, which shows only the presence of hypersecretion, congestion of food and accumulation of gas under the diaphragmatic roof in subdiaphragmatic abscess and perforating ulcer of the stomach. This is followed by examination with a contrast substance. The first step is to study the relief of the mucous membrane, for which purpose the patient is given 2-3 mouthfuls of barium. Examination of the folds of the mucous membrane may disclose slight changes escaping notice after large portions of contrast substance have been taken. Normally, the mucosal folds have a lengthwise arrangement, especially along the lesser curvature. In certain types of gastritis the folds become rough, while in anacidic gastritis they are mostly atrophic and obliterated. Examination of the mucosal relief is important for identifying tumours since in patients with malignant newgrowths the relief breaks off whereas in those with benign newgrowths the relief is intact and bypasses the tumour. After a study of the mucosal relief the patient is told to swallow the remaining suspension gradually, i.e., the examination is now continued with the stomach being filled up to repletion. The attention is turned to the position, form and tone of the stomach, peristalsis and the motor function.

Roentgenoscopy may help locate the position of the stomach. In gastroptosis the lower end of the stomach lies at the entrance to the small pelvis. Distention of the stomach is manifested by its considerable ptosis and a horizontal level of the contrast substance. In such cases, the stomach has the form of a brimming cup.

According to the condition of its muscles the stomach may be hypertonic, hypotonic or atonic. With an increased tone the stomach position is high, with a decreased tone it is low. In a hypertonic stomach a mouthful of barium suspension moves into the stomach slowly because of the strong resistance from the latter's walls. In a hypotonic stomach the stomach wall offers weak resistance to barium which takes the form of a long wedge, whereas in an atonic stomach barium immediately falls into its lower part as a result of the absence of any resistance.

Attention should be paid to displacement of the stomach in inhalation, forced expiration and in tension of the abdomen. The absence of such displacement is encountered with adhesions fixing the stomach.

Peristalsis may be normal, lively, sluggish, deep, and segmentary. Normal peristalsis is of average depth. It usually begins in the larger curvature at the entrance to the stomach and reaches the pylorus within 20 to 25 seconds on the average. In a lively peristalsis several wave-like movements are noted to follow one another along the lesser and the larger curvatures. In a sluggish peristalsis the waves are rare and superficial. In a deep peristalsis the waves slowly go down the stomach wall and sometimes divide the stomach into separate segments. In patients with inflammations or tumours peristalsis is absent in the affected areas.

The motor function of the stomach may be determined from the amount of barium suspension remaining in the stomach $1^1/_2$-2 hours after it has been taken. Normally, by this time the barium remainder should not exceed one-fourth of the suspension administered. Under normal conditions, evacuation of the stomach begins as early as a few minutes after swallowing the contrast mass. Evacuation at an earlier time is considered brief, and that at a later time, in 3-4 hours, slow. Detection of barium suspension in the stomach after 6 hours and later indicates a considerable delay in evacuation.

X-raying of the stomach serves to identify gastritis, as well as ulcer, cancer, polyps, and cicatricial changes in the stomach.

Examination of the duodenum. This examination is undertaken immediately after x-raying of the stomach. It is begun in the anterior (sagittal) projection and is continued in oblique projections all along the length of the duodenum. To locate a niche, aimed photographs are taken to show all the stages in the periodical filling of the duodenum. For a prolonged filling, the examiner compresses the descending portion of the duodenum. The niche is usually located in the posterior wall of the duodenal bulb, and less frequently in the lower part of the duodenum. Cicatrization of an ulcer of the duodenal bulb causes the latter's deformities of queer forms resembling a trifoil, a butterfly, etc. Sometimes, a considerable constriction of the duodenum occurs, which leads to distention of the stomach.

Examination of the large intestine. X-ray examination of the large intestine is carried out after introducing a contrast mass per os, injecting of contrast substance through an enema or injecting of air into the rectum through a balloon used to pump air visualizing the process on a screen. When a contrast mass is taken per os, the object of study is its movement through the intestine. Normally, the caecum is filled up in a matter of 4 hours, the right flexure of the colon in 4-6 hours, the left flexure in 9-12 hours, the sigmoid flexure in 12-15 hours, and the rectum in 14-20 hours. Evacuation of barium suspension from the intestine is completed in 24-48 hours. This method of examination may help determine the position of the intestine, discontinuity of tissue in its wall, and rough anatomical changes within it. To reveal slight changes, it is best to inject a contrast suspension through an enema. For this purpose, 500 g of barium sulphate is dissolved in 1 litre of lukewarm water and introduced into the rectum. After examining on the screen the character or degree of filling the intestine, its form, position and tone, the patient is told to evacuate the bowels, and the intestine is examined again. This time the mucosal relief is studied, since a certain part of the contrast substance is deposited on the mucous membrane after evacuation of the bowels.

X-ray examination using rectal injection of air is employed to determine the position of the large intestine with regard to other organs. Examination of the intestine may reveal lesions of the folds which evidence the presence of a severe inflammation. Slight inflammatory changes are not detectable by x-rays. A newgrowth in the large intestine is comparatively easy to diagnose.

Cancer of the large intestine can be detected even at an early stage from changes in the intestinal mucosa. Knobby tumours are most commonly confined to the rectum, the sigmoid, and the caecum and are revealed roentgenologically as a filling defect. A distention is found above the site of carcinoma. Constriction of the intestine is more often encountered in scirrhus (scirrhous carcinoma, fibrous cancer), which produces a circular constriction as though the intestine were tied up with a string. The clinical picture of intestinal polyposis is similar to that of cancer. Multiple polyposis produces a pattern of tracery. Tuberculosis of the caecum, particularly its tumorous forms, also gives a clinical picture similar to that of cancer. It is diagnosed by comparing x-ray findings with the clinical picture.

X-raying reveals various anomalies in the development of the intestine. An interesting phenomenon is elongation of the sigmoid with its simultaneous dilation, which is known as megacolon, and elongation of the transverse colon, resembling a double-barrelled gun.

Cholecystography enables examination of the form, position, and function of the gallbladder and the biliary ducts with the aid of a contrast medium injections and x-ray photography. X-ray examination of the gallbladder, without using a contrast medium, sometimes reveals only calculi containing lime. Therefore, the capacity of the body to excrete certain injected substances through the liver has long been used for x-ray examination of the gallbladder with the aid of bilitrast, tetragnost, iodognost (iodeikon).

Cholecystography is undertaken after preliminary preparations during which a patient is for three days given light food containing no coarse cellulose and substances likely to cause flatulence. The intestine is cleansed exclusively with purgatives. Two hours before injecting a contrast substance the patient is given 2-3 raw egg yolks to swallow so as to free the gallbladder from bile (egg yolk is known to be an effective cholagogue). Egg yolks may be substituted by a hypophysin injection. This is followed by administration of a contrast substance in tablets per os. The latter are dissolved in the alkaline medium of the duodenum. A contrast substance may also be injected intravenously in a dose of 0.05 g per kg of body weight, dissolved in 50 ml of water. No intoxication follows peroral administration of a contrast substance but the photographs thus obtained are less distinct. In recent time, cholecystography has been performed with a new preparation, biliselectan, which is perfectly harmless to health and gives a distinct x-ray picture. Photographs are taken once in every 6, 12, 18 and 24 hours. No gallbladder shadow is found after the application of a contrast substance in the following cases: (a) a weakening of the gallbladder capacity to concentrate bile and to contract; (b) obstruction of the bile duct; (c) inflammatory processes in the gallbladder; (d) diseases of the liver. Cholecystography also helps detect gallstones in the gallbladder.

Examination of the urinary organs. *A scout film of the kidneys* taken without a preliminary injection of a contrast medium shows only the lower two-thirds of the kidneys. The kidney shadow lies alongside of the shadow of the spine, somewhat outwardly to it. The ureters are not visible in the film. Calculi rich in lime salts may be seen sometimes. Such scout films give only a very general idea of the size and position of an organ and reveal the presence of calculi (although not all of them are detectable by x-rays) and tumours of the ureters. Uric acid calculi are poorly visible, while oxalates are seen best of all. Phosphate stones are quite unobservable.

An important role in the diagnosis of diseases of the kidneys and urinary passages is played by x-ray photography undertaken after injecting a contrast medium (pyelography, cystography). The contrast medium used in the Soviet Union is sergosin (skiodan).

In *pyelography* the contrast medium is introduced under the cystoscopic control into a ureter through a catheter, after which pictures are taken while the patient is holding his breath to avoid displacement of the organ. This method is inapplicable in cases of constriction of the urethra. A contrast medium injected intravenously is excreted from the blood into the lumina of the renal tubules, in which it concentrates and forms especially salient contours of the kidneys and their pelves as well as of the ureters and urinary bladder. Excretion of a contrast medium begins as early as in a quarter of an hour, and in three hours' time it is completely eliminated from the body.

In pathology, pyelography reveals changes in the size and form of the kidneys, renal pelves, and ureters as well as the presence of calculi. Pyelography is also used to detect kidney tumours invading a renal pelvis, because this is likely to produce in the x-ray film a defect with uneven, jagged contours (cancer, hypernephroma). In renal tuberculosis pyelography is employed to reveal cirrhotic changes, cavities, and calcarous deposits which occur during the organization of tuberculous foci and are sometimes mistaken for lime stones. The first picture is taken 5-10 minutes, and the second one 15-20 minutes after a sergosin injection into the blood stream. Roentgenographic studies of the urinary bladder are of no great practical importance because this organ can be inspected through a cystoscope. The latter is a narrow metal cylinder provided with an optic system similar to that of the microscope. It is used to inspect the mucous membrane of the bladder and the outlets of the ureters. Urine can be taken with a catheter from each of the ureters separately under cystoscopic control so as to ascertain the functional condition of each of the kidneys.

ELECTROCARDIOGRAPHY

Electrocardiography as a method of examination was invented about fifty years ago. It is based on the principle of recording the electromotive variations in the action of the heart muscle. Each section of the heart, when in a state of stimulation, is electrically negative with regard to the section at rest. This accounts for a difference between the electrical tension of different sections of the heart.

Action potentials can be recorded in any part of the body. The extremely slight difference in voltage observed in this case is detected with the aid of a highly sensitive galvanometer, which is made as a small strained steel plate with a glued-on mirror in a strong electromagnetic field. A focussing lens is set in front of the mirror. Changes in the magnetic field, occurring under the influence of action potentials, cause rotation of the plate with its glued-on mirror and deflection of the ray reflected from the mirror to one side or the other. Vibrations of the plate are registered as curves in a photograph.

The time marker is a tuning-fork tuned to 20 vibrations per sec which gives vertical deflections at intervals of 0.05 sec.

The recording unit of the apparatus consists of a delivery casette and a chart-winding mechanism operating like a gramophone. The recording is made on a perforated chart or photographic film.

An electrocardiogram (ECG) must be taken in a warm room, so that the patient will not have an unpleasant sensation of cold and will not shiver. He should lie quietly on a wide bed or couch. To eliminate external currents, a special iron net is placed under the blanket and grounded with a wire connected to a water-pipe or heating system. Before the recording procedure, one is obliged to check upon the power supply to the electrocardiograph, and the sensitivity of the measuring system which should ensure a deviation of the beam reflection by 10 mm from the isoelectrical line.

The electrical potentials of the heart are projected in various points on the body surface and recorded by the electrocardiograph. They may be led off the following three points on the body: the right arm, the left arm, and the left leg. The electrode of the right arm is connected to the wire of the negative pole of a galvanometer, the left arm and the left leg to the wire of the positive pole. The difference between the potentials of the right and the left arms is identified as lead I, the right arm and the left leg as lead II, the left arm and the left leg as lead III. These are called standard leads. For a more elaborate diagnosis other leads are also used. Precordial leads are considered to be the most common. One is advised to keep the electrode of the right arm turned on while the electrode of the left arm is set in the following positions: at the right border of the sternum in the fourth intercostal space—precordial lead I, at the left border of the sternum in the fifth intercostal space—precordial lead II, in the middle between the left border of the sternum and the midclavicular line in the fifth intercostal space—precordial lead III; midclavicular line in the fifth intercostal space—precordial lead IV; the anterior axillary line in the fifth intercostal space—precordial lead V; the median axillary line in the fifth intercostal space—precordial lead VI.

The electrical phenomena occurring in the heart, when registered by an electrocardiograph, give a characteristic curve with three waves, *P*, *R*, and *T*, directed upwards, and two waves, *Q* and *S*, directed downwards. The *P* wave reflects the process of stimulation of the atrium. The *P-Q* interval corresponds to the conveyance of an impulse from the sinus ganglion to the atrioventricular node, and is equal to 0.12-0.18 sec; *QRST* reflects the process of stimulation and contraction of the ventricles; *QRS* reflects the primary stimulation at different points in the right and left ventricles, which lasts 0.06-0.08 sec.

Most authors attribute the *T* wave to the process of cessation of stimulation in the ventricles. The time of ventricular systole *QRST* is 0.32-0.35 sec. The *T-P* interval is the cardiac pause lasting 0.27-0.32 sec (Fig. 11).

In hypertrophy of the left ventricle, there is a deep *S* wave in lead III—the sinistral type of an electrocardiogram. In hypertrophy of the right ventricle, there is a deep *S* wave in lead I—a dextrogram. Since the *P* wave reflects the action of the atrium, a change in its form and size is used for judgement on the presence of pathologic processes in the atrial myocardium. Prolongation of the *P-Q* interval (over 0.20 sec) may result either from an increase in the vagus nerve tone or from an anatomic dislocation in a given portion of the conducting system.

A deep *Q* wave (over 4 mm) is evidence of a local change in the ventricular myocardium, especially in the presence of a negative *T* wave; the Q_{I} wave of a former infraction of the anterior wall; the Q_{III} wave of a scar in the posterior wall.

A change in the size and form of the *R* wave is seen with hypertrophy of the ventricles, arrhythmia and coronary insufficiency. Deviation of the *S-T* interval below the isoelectrical line is due mainly to a change in the myocardium as a result of coronary circulation disturbance. A change in the size and form of the *T* wave evidences a disorder in ventricle contraction.

Electrocardiography helps one estimate changes in the basic functions of the heart: impulse generation excitability, conductivity and contractability. Therefore, this method is of certain value for a study of the normal and pathologic rhythm of the heart's action. ECG data may aid in specifying the character of acute affections of the myocardium by demarcation of focal and diffuse changes. Electrocardiography is particularly important in acute disturbances of the coronary circulation, thrombosis of a branch of the coronary artery (infarction), and in various forms of arrhythmia.

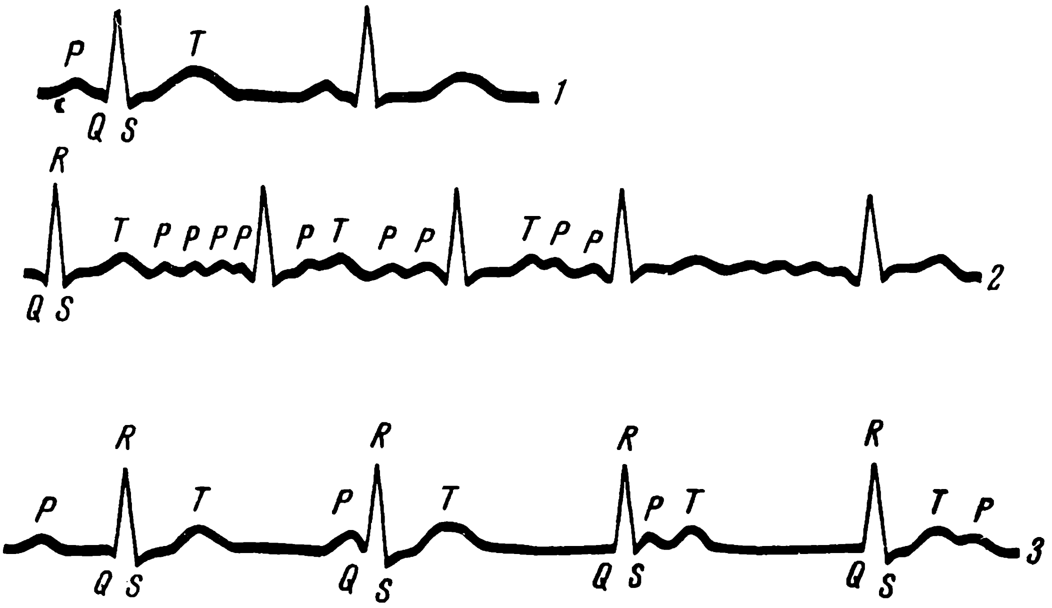


Fig. 11. Electrocardiograms

1—normal electrocardiogram; *2*—electrocardiogram in atrial fibrillation; *3*—electrocardiogram in transverse block

Examination of the Sputum

Healthy persons do not discharge any sputum. A small quantity of phlegm is secreted by smokers. For judgement on its character, the sputum expectorated in 24 hours is collected in a clean dry vessel and measured. Either one or two spittles collected in the morning on awakening or the entire quantity of sputum expectorated in 24 hours may be examined. Sputum collected in 24 hours is preferably kept in a dark cool vessel covered with a lid. The samples are examined macroscopically and microscopically so as to determine the colour, odour and quantity of the sputum, its stratification and character, as well as various inclusion bodies found within it.

Sputum may be of a liquid, mucous, watery, viscid and tenacious consistency.

Newly-discharged sputum usually emits no odour. In pulmonary gangrene, it is foetid.

The colour and transparency of sputum depend on its character. Mucous sputum is colourless and transparent. Mucopurulent and purulent sputum is of a turbid yellow or greenish colour. Rusty sputum occurs with lobar pneumonia. Brown sputum is the result of decomposition of haemoglobin and formation of haematin, and is seen with pulmonary gangrene. Sputum of a raspberry jelly colour is encountered in lung cancer. It may turn black after inhalation of coal dust.

Two-layered sputum is characteristic of pulmonary abscess. In pulmonary gangrene the sputum has three layers: the upper, frothy layer with mucopurulent scraps hanging down into the middle layer; the middle, liquid layer; and the lower, loose, flaked layer consisting of detritus and pus corpuscles.

Sputum may contain mucus, pus, and sometimes serous fluid. As to its *character*, sputum may be mucous, serous, purulent, and sanguineous, or contain these components in various combinations. Mucous sputum is secreted in the early days of catarrhal conditions of the upper air passages; viscid mucous sputum in lobar pneumonia; glassy sputum in bronchial asthma; purulent sputum in pulmonary suppurations (abscess, bronchiectasis, etc.). Mucopurulent sputum is characteristic of most diseases of the bronchi and lung parenchyma.

Sanguineous sputum and blood-spitting (haemoptysis) are encountered in pulmonary tuberculosis, bronchiectasis, syphilis and lung cancer. Serous sputum is seen mainly with pulmonary oedema.

Macroscopic examination of sputum. Sputum spread in a thin layer over a Petri dish is examined with the naked eye against a black background (black paper). This may reveal the presence of a number of inclusion bodies of diagnostic importance, such as (a) Dittrich's plugs—whitish pellets the size of a millet consisting of bacteria, products of cell decomposition, and fatty acid crystals. They have a foetid odour and are found in the lower layer of sputum in pulmonary gangrene and bronchiectasis; (b) arborescent clots of fibrin with a branchlike structure, which consist of mucus and fibrin. When present in quantity, they may obstruct the lumen of a bronchus. They are found in sputum of patients with fibrinous bronchitis and less often with pneumonia; (c) Curschmann's spirals—long coiled filaments occurring mainly in bronchial asthma; (d) material left by ruptured hydatid cysts; (e) pieces of a lung tumour are discoverable during disintegration of the newgrowth.

Microscopy of sputum. The objects of examination are both fresh, unstained (native) sputum and sputum stained by special methods. For examination of fresh, unstained sputum lumps of the sputum are transferred with a heat treated needle from a Petri dish to a clean slide. A lump is slightly pressed with a cover-glass so that the sputum is spread in a thin layer and none of it is squeezed out from under the cover-glass. Normally the preparation may be found to have desquamated cells of the squamous epithelium lining the respiratory passages. These are large, polygonal cells having small nuclei. A sputum preparation may contain erythrocytes, leucocytes, Curschmann's spirals, Charcot-Leyden crystals, tyrosine, fatty acids, Dittrich's plugs, parasites, and bronchial clots.

Cylindrical cells of the ciliated epithelium are found in sputum from patients with bronchitis. These cells are oblong and have an elongated end. In pulmonary tuberculosis and a number of other lung diseases sputum may contain cells of the alveolar epithelium (round cells with a large round nucleus). Isolated erythrocytes and leucocytes occurring in the field of vision are also encountered in the sputum of healthy persons.

The presence of a considerable number of erythrocytes in sputum is certain evidence of a disease. Erythrocytes are present almost in any sputum, but their number in sanguineous sputum is considerably above normal. They are easily recognized by their colour and shape (round nuclear-free cells). Erythrocytes of pulmonary origin are uniformly mixed with sputum. In a sputum sample they sometimes appear as colourless circles, the so-called shadows, and are evidence of a former or continuing slight haemorrhage. Often erythrocytes disintegrate in sputum, and cannot, therefore, be seen in microscopy. Nevertheless, the sputum is stained red by the liberated haemoglobin.

Leucocytes are also found in any sputum, but they are particularly numerous in purulent sputum. The latter entirely consists of leucocytes which may be undergoing various stages of degeneration. Sometimes sputum is found to contain only the nuclei of leucocytes left after the death of the frail protoplasm. In some cases sputum contains only detritus, i.e., a mass of tiny granules, decomposition products of leucocytes.

In patients with heart defects, particularly mitral stenosis accompanied by congestion in the pulmonary circulation, brown-coloured cells of the alveolar epithelium appear in sputum. Their colour is due to the presence of iron absorbed from the haemoglobin of erythrocytes. They are known as heart-disease cells.

Charcot-Leyden crystals are found in combination with Curschmann's spirals in bronchial asthma. Curschmann's spirals appear under the microscope as twisted, delicate filaments. Charcot-Leyden crystals are lustrous, transparent octahedrons. They are formed of decomposition products of eosinophils.

Elastic fibres are discovered easier after special treatment of sputum. Equal quantities of sputum and a 10 per cent caustic potash solution (5 ml each) are mixed and heated in a test tube over the flame of a burner until the entire mass has become homogeneous. In this way all the constituent parts of sputum are destroyed, and only the elastic fibres remain intact. After centrifugation of the fluid, a preparation made from the sediment may be found to contain elastic fibres with distinct double contours divided in two at the ends. When stained with a 1 per cent alcohol solution of eosin they are even better visible.

Bacterioscopic examination of sputum. For making a preparation a lump of sputum is transferred on a heat-treated needle to a slide and covered with another slide. After the sputum has been uniformly spread between the slides, the latter are separated by sliding them one upon another. Both preparations are dried and fixed by their triple passage, smears upwards over the flame of a burner. They are stained as follows: (a) with fuchsin; (b) by Gram's method; (c) by Ziehl-Neelsen's method.

For *staining with fuchsin*, an aqueous solution of carbolic fuchsin, 1 : 10, contained in a drop-bottle is poured on a fixed smear through a pipette, allowed to remain for 2-3 minutes, and washed off with a stream of water. The smear is then carefully dried and placed between two sheets of filter paper for further drying.

When *staining by Gram's method*, a piece of filter paper, somewhat smaller in size than the slide, is placed upon a fixed preparation, whereupon a carbolic gentian violet solution is poured over it. Filter paper protects the smear against stain deposition. In 2-3 minutes' time, the paper is removed with a pincers, the stain drawn off, and Lugol's solution poured on the smear as a mordant to be also drained in 1-2 minutes. The slide is put in a glass filled with 192 proof alcohol, shaken during a few seconds, and washed with water. Should its discoloration be incomplete, the procedure with its immersion in alcohol is repeated until the smear has turned grey, after which it is stained with fuchsin during 1-2 minutes, washed with water and dried. By this stain some bacteria are coloured blue, while others take a pink colour. Bacteria which stain blue are called gram-positive, while those staining pink are known as gram-negative bacteria.

The following bacteria are stained by Gram's method: (1) pneumococci, or Fränkel's diplococci, which are encapsuled organisms. Their adjoining ends are rounded, while the opposite ends are pointed, resembling a candle flame and having a light rim (capsule). These pneumococci are gram-positive and occur mainly in the sputum of patients with lobar pneumonia, but may also be encountered in those with bronchopneumonia; (2) pneumobacteria, or Friedländer's diplobacteria, which are rod-shaped encapsulated organisms with rounded ends. They are gram-negative and occur in patients with lobar pneumonia. Staphylococci, streptococci, Pfeiffer's bacilli (oval rods), spirochaetes, and spindle-shaped bacilli (*Bac. fusiformis*) are gram-positive.

Mycobacterium tuberculosis is stained after Ziehl-Neelsen. These organisms are acid- and alcohol-fast, and are, therefore, stained by special methods. Ziehl-Neelsen's method is as follows: A piece of filter paper, not larger in size than the slide, is placed on a dried and fixed smear; undiluted carbolic fuchsin is poured over its surface, and the slide is kept over the flame of a burner until vapours appear. While doing so, caution should be displayed lest the stain dries up, therefore, more stain is added from time to time. After this the preparation is left alone for 2-3 minutes until it has cooled down somewhat. The paper with fuchsin is removed, the smear is washed with water and immersed for one minute in a glass of 5 per cent sulphuric acid solution. It is then thoroughly washed with water, counterstained with a methylene blue solution, 1 : 1,000, during 2-3 minutes, again washed and dried. The stained smear is examined through a microscope with an immersion system, and is found to contain mycobacteria of a red colour, which are thin and sometimes curved. They may be few in number or as numerous as to cover the entire field of vision. Mycobacteria phagocytized (ingested and destroyed) by leucocytes may also be observed. The smear is otherwise coloured blue throughout (Fig. 12). One should prepare 3-4 smears from one and the same sputum simultaneously. In addition to smears of ordinary sputum, concentrated sputum containing an increased number of *Mycobacteria tuberculosis* is also examined. This concentration is effected as follows: (a) by treatment of sputum with antiformin, which destroys all organisms save for *Mycobacteria tuberculosis*; (b) by flotation; (c) by inoculation of sputum into special media; (d) by infection of animals; (e) by examination of a patient's gastric juice.

Sterilization of sputum after its examination is obligatory. It is sterilized by autoclaving or exposure to various disinfectants.

Examination of Membranes

The material from the patient's throat is collected on a swab (sterile cotton-wool tampon). There are several methods for detecting diphtheria bacilli: (a) examination of a smear; (b) an express method; (c) cultivation on special media.

A smear is prepared from material taken on a swab from a patient's fauces or nasal cavity. The swab is rubbed over heat-treated slides, the smears produced thereby are fixed in the flame of a burner stained with Loeffler's methylene blue or Stoltenberg's stain during 2-3 minutes, and examined by microscopy. A negative result of this investigation is no guarantee of the absence of diphtheria bacilli. If the latter are discovered, however, the diagnosis is made a day earlier, which is very important for timely diagnosis and treatment.

Formulas of dyes. 1. Stoltenberg's stain: methylene green 2.5 g, toluidine blue 2.5 g, concentrated acetic acid 100 ml. The stain is oxidated by adding 20 drops of a saturated solution of potassium bichromate and simultaneous trituration, after which its volume is brought up to 1 litre with distilled water.

2. Loeffler's alkaline methylene blue: distilled water 100 ml, 10 per cent potassium hydroxide solution 2 drops, 7 per cent methylene blue solution 30 ml (or methylene blue 3 g), 192 proof alcohol 100 ml, 1 per cent potassium hydroxide solution 1 ml.

The express method enables detection of diphtheria bacilli in 4-5 hours' time.

A sterile swab soaked in horse serum is put into a test tube (to speed up coagulation serum is kept in a water bath at a temperature of 80-90°C for half-an-hour). Material is then collected from the fauces with the swab and kept in a thermostat at a temperature of 37°C for 4-5 hours. A slide smear is made as usual and stained by a single-stage procedure with Loeffler's or Stoltenberg's stain or by Neisser's two-stage method and subjected to microscopy. One need have a certain experience to be able to discover characteristic granules at the ends of a diphtheria bacillus. In case the test results are negative the same swab is used for implantation of smear samples in nutrient media.

Staining by Neisser's method is carried out twice: once with Neisser's dye and with Lugol's solution during 1 minute, after which the preparation is washed with water and contrastained with chrysoidine or vesuvin, otherwise known as Bismarck brown, during 1-3 minutes and again washed with water.

The composition of dyes. 1. Neisser's dye: methylene blue 0.1 g, 192 proof alcohol 2 ml, glacial acetic acid 5 ml, distilled water 100 ml.

2. A solution of chrysoidine or vesuvin (Bismarck brown) 1 g, distilled water 300 ml. The dye is dissolved in heating water and filtered.

Inoculation into nutrient media. Material collected from a patient's fauces is implanted in Loeffler's serum or coagulated blood serum contained in a test tube. The swab should be rubbed into the nutrient medium on all sides. Whenever a culture medium is unavailable at the time the material is collected, one is advised to use a swab moistened with a physiological salt solution or a 15 per cent glycerol solution and keep it in a stoppered sterile test tube. The time interval between taking the material and its inoculation into a culture medium should not be longer than 5-6 hours.

It is recommended to take material from the fauces and nose simultaneously on different swabs to be put into different test tubes. An inoculated culture medium is kept in a thermostat at a temperature of 37°C. In 18-20 hours' time the test tubes are taken out. Diphtheria bacilli growing in a culture medium form numerous string-shaped, densely arranged colonies on its surface or lend it a characteristic shagreen appearance.

Preparation of a smear from a grown pure culture. Material from a grown colony is taken with a loop and introduced into a drop of physiological salt solution on a clean slide, whereupon a thin smear is made, dried in the air, fixed over flame and stained with Loeffler's methylene blue and Stoltenberg's stain. Two or three smears are thus prepared. If diphtheria bacilli are present they are seen among the common flora as aggregations looking like felt.

These bacilli vary in form. They may be pointed or thickened at the ends, often arranged at an angle to one another, and have at their ends characteristic dark granules which are a reserve nutrient. These granules are better visible when stained by the Neisser's method, the bacilli taking a brown colour and the granules a dark-blue one. Neisser's staining method is used in doubtful cases.

Convalescents after diphtheria and their human contacts may long remain *bacilli carriers*, the source of a certain epidemiologic danger. Bacilli carriers are discovered by a study of cultures of material taken from the fauces and nose, but such a culture is kept in a thermostat for 48 hours to facilitate the germination of bacilli.

Blood Tests

Laboratory investigation of the morphology of the blood is very important for diagnosis and prognostication, and today is obligatory for the examination of every inpatient and many outpatients.

A morphologic examination of the blood includes: (1) determination of the percentage of haemoglobin; (2) red-cell count; (3) determination of the colour index; (4) white-cell count; (5) differential count of white cells, i.e., calculation of the percentage of individual types of leucocytes; (6) erythrocyte sedimentation reaction (ESR), and sometimes, a thrombocyte count.

Technique of taking a blood sample. Most commonly, a blood sample is drawn from the tip of the fourth finger. The finger tip is carefully wiped with 192 proof alcohol and dried with cottonwool, slightly pressed at the base of the nail phalanx and punctured with Francke's needle. The latter's convenient design enables adjustment of puncture depth by turning the needle head and drawing its blade to a required length according to skin thickness. The exposed part of the needle blade must be at least 3-5 mm in length, since a shallow puncture will not yield sufficient blood and requires finger pressure, which causes intense pain and changes the conditions of investigation to a degree where another puncture may be necessary. A Francke needle should be thoroughly wiped with alcohol before use. One puncture is enough to draw sufficient blood to make study of haemoglobin, erythrocytes, leucocytes, ESR, and thrombocytes.

Determination of the percentage of haemoglobin. The first drop of blood is removed with a piece of cottonwool, and the second is drawn into a Sally's capillary tube up to the mark for estimating haemoglobin. Caution should be displayed so as not to draw superfluous blood and avoid air bubbles. Therefore, as soon as the blood level has reached the mark, the rubber tube with which Sally's capillary tube is provided is compressed with the fingers, the end of the capillary tube is wiped dry with cotton, and the blood level checked again. The capillary tube is then inserted into a Sally's test tube filled with a decinormal solution of hydrochloric acid up to the 10th graduation so as to have only its end slightly submerged in the acid, whereupon the blood is carefully blown out into the test tube. The last drop of blood is blown out not into the acid but above its level onto the wall. The test tube is then tilted to wash the drop off the wall. The fluid rapidly, in fact, before the examiner's eyes, changes from red to brown of variable intensity depending on the percentage of haemoglobin. It is once more drawn into the Sally's capillary tube so as to wash the blood completely off the walls, is again filled with liquid whereupon one has to note the time and place the test tube in a glass packed with cottonwool. Haemoglobin is estimated by the colorimetric method, i.e., by means of comparing the colour of a standard haemoglobin solution with that of haematin hydrochlorate already formed in the hydrochloric acid.

The haemoglobinometer (Fig. 13) consists of a wooden frame with three sockets. The two side sockets accommodate test tubes filled with standard solutions, while the middle socket is intended for a graduated test tube containing a solution of haematin hydrochlorate subject to investigation. The colours of these solutions have to be compared. If the solution in the middle test tube is paler than that in the standard ones, the same amount of blood is again drawn

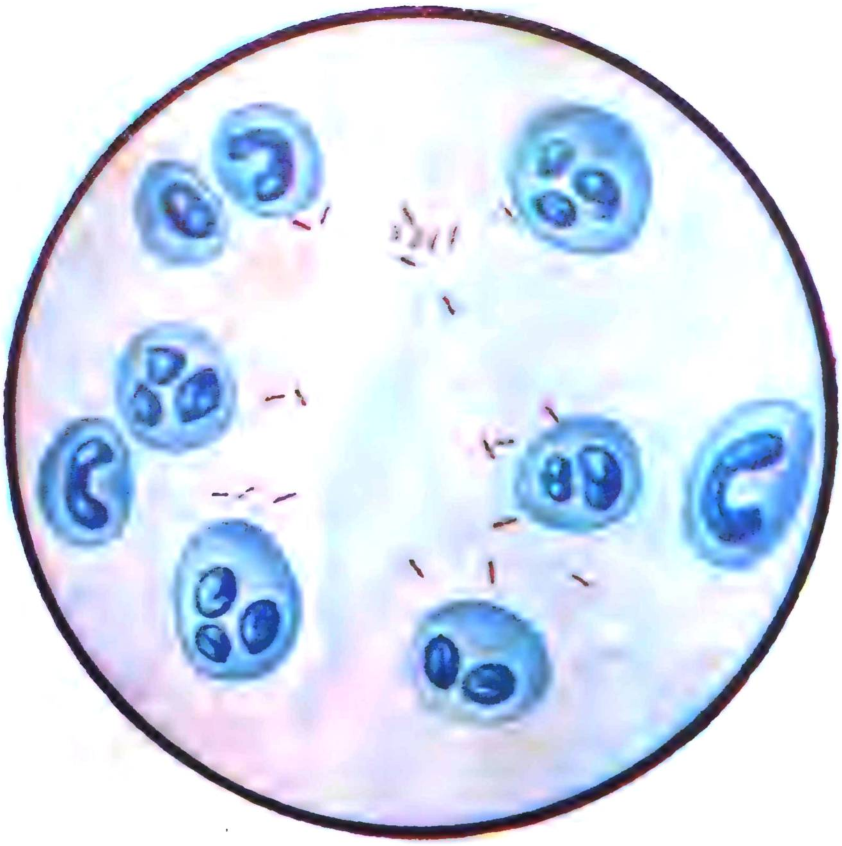


Fig. 12. *Mycobacterium tuberculosis.* Stained after Ziehl-Neelsen's method

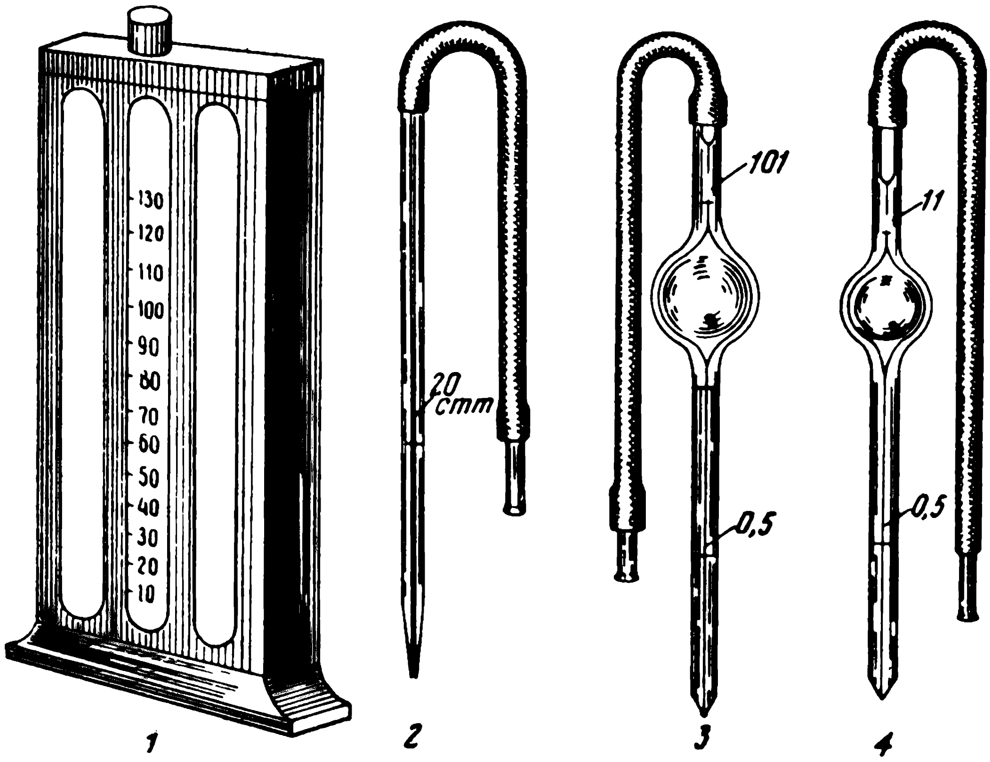


Fig. 13.
1—haemoglobinometer; 2—pipette for taking blood; 3—mélangeur for erythrocytes; 4—mélangeur for leucocytes

from the finger, and the result obtained is divided by half during calculation. If, however, the solution under examination is darker than the standard solution, colorimetry is begun in 10 minutes' time. For this purpose, the haemoglobinometer is taken in the left hand and kept on a level with the eyes, the right hand being used to add distilled water through a medicine-dropper to the solution examined, which is then stirred with a glass stick. Water is added until the contents of the middle and lateral test tubes become uniform in colour. The divisions on the graduated test tube indicate the haemoglobin content of the blood under examination. The level of the solution in the graduated test tube having the same colour as the standard test tubes will indicate the percentage of haemoglobin in the blood under investigation. Estimation of haemoglobin permits an error of 5 per cent. At present, the haemoglobin content of the blood is also expressed in g% units. The normal haemoglobin content is 16 g% in 100 ml which corresponds to 100 per cent of haemoglobin.

Blood sampling for a red cell count. From the same puncture in the finger the next drop is drawn into a red cells mixer (mélangeur), which is a graduated capillary pipette with a bulb-shaped expansion (reservoir) containing a red-coloured glass ball (Fig. 13). The blood is accurately drawn up to the mark 0.5 and, in the presence of anaemia, to mark 1. After this a 0.85-1 per cent common salt solution is drawn into the mélangeur to the mark 101, taking precautions against an escape of blood. If blood is collected up to the mark 0.5, and the volume is brought up to the mark 101 by adding diluting

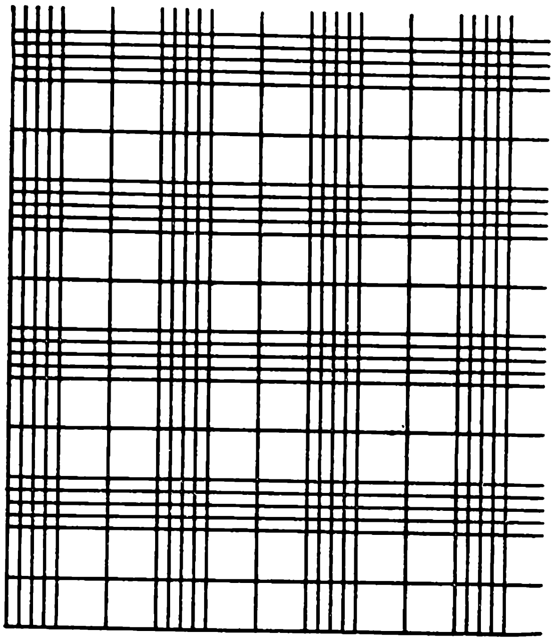


Fig. 14. Grid of Goryayev's counting chamber

fluid, the blood dilution ratio will be 1 : 200; if blood is collected up to the mark 1, the dilution ratio will be 1 : 100.

The capillary tube maintained in a horizontal position is compressed between two fingers and shaken during 2-3 minutes, after which a drop of diluted blood is transferred into a counting chamber. It should be remembered that the first 2-3 drops of fluid are unfit for a cell count and must be discarded.

Obtaining a blood sample for a white cell count. For counting the leucocytes a drop of blood is taken from the same puncture into a mélangeur specially adapted for this purpose and having a white ball in the ampule with the mark 11 above the latter (Fig. 13). Leucocytes are diluted with a 3 per cent acetic acid solution which dissolves erythrocytes. To make the leucocytes better visible, 2-3 drops of a 1 per cent gentian violet aqueous solution are added to the acetic acid for staining leucocyte nuclei. The blood is drawn into the mélangeur up to the 0.5 mark, whereupon the acetic acid solution is sucked in up to the 11 mark, i.e., the blood is diluted 1 : 20. With a large number of leucocytes the blood is drawn into a mélangeur used for a red blood count up to the 0.5 mark (1 : 200 dilution) and shaken in the same way. The first 2-3 drops unfit for counting are poured off.

The counting chamber. There are several models of counting chambers. In the Soviet Union, the most commonly used is Goryayev's chamber (Fig. 14). The counting chamber is made of a thick glass plate on which is engraved a special grid—a square surface divided into 225 large squares by a number of lines. Each large square is in its turn subdivided into 16 smaller squares. The side of the smaller square measures 1/20 mm, while its area is equal to 1/20 × 1/20 = 1/400 sq mm. The grid lies 1/10 mm below the slide surface. Thus, if a cover glass is pressed firmly to the surface of the chamber glass, there will be a slit 1/10 mm in height between the grid surface and the lower surface of the cover glass. What we call the counting chamber is in fact the space of this slit limited by the grid.

Since the area of the small square is equal to 1/400 sq mm, and the chamber depth is 1/10 mm, the volume of the small square is, consequently, equal to 1/400 × 1/10 = 1/4,000 cu mm. Knowing the number of blood cells contained in one smaller square (i.e., in 1/4,000 cu mm), it is easy to calculate their number per cu mm.

Before making a count, a polished cover glass is placed on the washed and dried chamber and carefully ground into the surface of the chamber glass (until the appearance of opalescent circles). A drop of fluid from the mélangeur is put into the slit formed between the grid surface and cover glass. The chamber thus filled is placed on the stage of a microscope, allowed to remain for 2-3 minutes to settle the blood corpuscles whereupon the count is begun. It is carried out at low magnification rate with a lowered condenser and a slightly covered diaphragm.

The red cell count. The erythrocytes are counted in five large squares or, which is the same thing, in 80 small squares. Let us presume that 80 small squares contain 400 erythrocytes; then, each square will contain 400 : 80 = 5. Since the volume of one small square is 1 : 4,000 cu mm, the number of erythrocytes per cu mm will be 4,000 times larger, i.e., 5 × 4,000 = 20,000. Since the blood introduced into the chamber was diluted 1 : 200, the actual number of erythrocytes per cu mm will be 200 times larger, i.e., 20,000 × 200 = =4,000,000.

Practically, the red cell count is made in a much simpler way, viz., the number of erythrocytes is counted in five large squares, and the sum found is multiplied by 10,000.

The white cell count. The leucocytes are counted in 100 large, usually blank squares (not divided into smaller ones). The sum found is divided by 1,600 (100 large squares contain 1,600 smaller ones) and multiplied by 4,000 and by 20 (blood dilution in mélangeur). Practically, the following method is used: the number of leucocytes found in 100 large squares is divided by 2, and the quotient is multiplied by 100.

Preparation of a blood film. Films are prepared on clean slides freed from grease which are stored in a glass vessel filled with alcohol. The slide is wiped dry with a clean cloth, care being taken not to touch its surface with the fingers. With the same precautions the edge of the slide is brought into contact with the drop of blood on the finger, taking care that the slide surface does not touch the finger skin. After this, the slide is taken between the thumb and index finger of the left hand so that the drop of blood is close to the index finger. Then, a polished cover glass is placed with its narrow edge on the slide at an angle of 45° to the left of the drop and carefully brought to the latter. When the drop of blood has spread over the edge of the cover glass the latter is quickly and smoothly moved in the direction of the thumb. A good film ought to be thin, even, and have a velvety surface. One is recommended to prepare two films.

A film dried in the air is immersed in a bath containing methyl alcohol for 3 minutes, then taken out, by the unsubmerged edge with pincers and placed vertically on filter paper. Fixation in methyl alcohol may be replaced by fixation in a mixture of equal parts of alcohol and ether (45-60 min). A fixing fluid stored in a jar is usable for an indefinite period of time.

Film staining with the Romanowsky-Giemsa stain. The stain is a mixture of azure and eosine. It is dispensed ready for use. Two drops of stain per mm of distilled neutral water are taken. The stain is poured in a thick layer over a fixed film and allowed to remain for 40 minutes, whereupon it is drained and the film washed with water and dried.

A film thus prepared is examined under a microscope with an immersion system, a raised condenser, and a fully-open diaphragm.

When making a differential white cell count, all leucocytes getting into the field of vision should be registered. The leucocyte forms, such as basophils, eosinophils, juvenile, rod neutrophils and filamented neutrophils, lymphocytes, and monocytes, are registered separately. When 200 cells have been counted, the counting is stopped, and the percentage of each type of leucocytes is determined. The differential white cell count is made on four sides of a film to forestall an accidental error.

Preparation of a thick film. The thick film method is used to detect *Plasmodium malariae.* A thick film should be taken irrespective of an attack because the parasites may invade the peripheral blood in the period between attacks also. The blood is collected from a punctured finger onto a slide, just as in the preparation of a conventional film, or a drop is formed with an angle of another slide and slightly spread out. Two or three drops are formed on one slide and allowed to dry in the air with the slide in a horizontal position, whereupon they are stained without preliminary fixation with Romanowsky-Giemsa stain during 35-40 minutes. The remainder of the stain is carefully washed off with water, and the preparation is dried in a vertical position, after which it is ready for microscopy.

Most commonly, the parasites are found to form a circle inside the erythrocytes, but other stages of *Plasmodium malariae* may also be seen. Identification of individual types of parasites in a thick drop is difficult, and should better be undertaken in a film which is prepared in the same way as that used for determining the blood morphology.

The colour index. The colour index is the ratio of the haemoglobin content to the number of erythrocytes, i.e., it indicates the amount of haemoglobin contained in each red blood corpuscle. To calculate the colour index, a proportion is established between the relation of discovered haemoglobin to 100 per cent of haemoglobin and between the discovered number of erythrocytes to the normal red cell count, i.e., to 5,000,000.

Example: $\frac{60}{100} : \frac{4,000,000}{5,000,000} = \frac{60 \times 5,000,000}{100 \times 4,000,000}$

Cancellation of fractions gives the following result:

$$\frac{3 \times 5}{5 \times 4} = 3/4 \text{ or } 0.75$$

The colour index may also be determined in a simpler way. The amount of haemoglobin discovered is divided by the first two figures of the erythrocyte count multiplied by 2. If the number of erythrocytes is less than 1,000,000 only one first figure is taken instead of the two.

Determination of erythrocyte sedimentation rate by Panchenkov's method. If blood in which coagulation has been prevented by one method or another is placed in a vessel it will form two layers when allowed to settle. The upper layer will consist of the plasma, the lower of the erythrocytes. The time required for the erythrocytes to settle on the vessel bottom is known as the erythrocyte sedimentation rate (ESR). The ESR is determined in Panchenkov's apparatus. It consists of a frame on which 4-5 pipettes are fastened by means of special clamps. Each pipette is graduated from 0 to 100. At the graduation 50 there is the mark "S" and at the graduation 100 the mark "B".

The ESR is investigated in the following way. A clean dry pipette is washed out several times with a 5 per cent sodium citrate solution. Then the latter solution is taken in up to the mark "S" and let out into a watch glass. Further, the blood is twice drawn from a finger puncture up to the mark "B" and both times is carefully—so as to avoid the formation of bubbles—let out into the watch glass containing the sodium citrate solution. The mixture is cautiously and thoroughly stirred and taken into the same pipette up to the 100 graduation. The pipette is fastened in the frame in a strictly vertical position and allowed to stand. Exactly an hour later the level of the column of settled erythrocytes is registered. In healthy persons the ESR varies from 4 to 12 mm/hr.

Blood platelet (thrombocyte) count. A drop of a 14 per cent magnesium sulphate solution is put on a finger. Then, the skin of the finger is punctured through the drop with a needle. The blood effused is mixed with magnesium sulphate by stirring with an angle of a slide, and films are made from the resulting drop. The films are dried, fixed in alcohol and stained with the Romanowsky-Giemsa stain. The staining lasts 2-3 hours. The stained film is thoroughly washed with tap water and dried. The counting is carried out through the immersion-lens of a microscope. One has to count 1,000 erythrocytes and note the number of thrombocytes encountered during the latter procedure.

To determine the number of thrombocytes, one has to ascertain the number of erythrocytes per cu mm.

Example. The number of thrombocytes counted per 1,000 erythrocytes is 56. The number of erythrocytes per cu mm is 4,500,000.

If the number of thrombocytes per 1,000 erythrocytes is 56, that per 4,500,000 will be as follows:

$$X = \frac{4,500,000 \times 56}{1,000} = 252,000$$

The normal number of thrombocytes per cu mm is from 250,000 to 350,000.

Staining and counting reticulocytes. In certain diseases it may be necessary to count the number of reticulocytes. The staining is made with a 1 per cent alcohol solution of brilliant cresyl blue (solution is based on absolute alcohol). Smears of a drop of brilliant cresyl blue dye are prepared on thoroughly washed slides and dried. These smears are fit for long storage. A thin blood film is made on a stain-covered slide and immediately, while it is moist, put for 7-10 minutes into a dampening chamber kept at hand.

The blood film taken out of the chamber and dried, one begins to count reticulocytes with the aid of an immersion-lens microscope. For added convenience in calculation one is advised to narrow the field of vision, for which purpose a piece of paper with a triangular opening is placed into the eye-piece. The next step is to count 1,000 erythrocytes and register the number of reticulocytes. Normally, there are 5-10 reticulocytes for every 1,000 erythrocytes. Reticulocytes are erythrocytes containing a delicate, tender network (reticulum) arranged all over the corpuscle or forming a thick skein in its centre. The reticulocyte granules stain blue. Increased reticulocytosis is a sign of erythropoietic hyperfunction of the bone marrow.

Analysis of the Urine

The properties of the urine vary in connection with different factors: rest or vigorous activity of the body, environmental temperature, fluid intake, dietary pattern, etc. Therefore, urine for analysis and subsequent comparison of its results is taken under approximately identical conditions: on an empty stomach, immediately on awakening in the morning. The bed-pan must be clean, and the urine is poured off into a clean bottle with a label indicating the patient's full name, ward, and, whenever possible, the character of investigation. The bottle capacity must be at least 200 ml. The urine should contain no foreign admixtures; therefore, a woman's urine is taken preferably with a catheter after she has washed herself. During the menstrual period urine is not collected. The urine should be kept in a dark cool place. It is desirable to make its analysis not later than 2-3 hours after urination.

Quantity. The urinary secretion during 24 hours amounts to 1,200-1,500 ml, and is subject to considerable variations. It is believed that urinary secretion of less than 700 ml or more than 3,000 ml per 24 hours is evidence of a disease. The quantity of urine passed in 24 hours is collected in the following way. On rising in the morning the patient is told to void urine; all subsequent portions of urine passed during 24 hours are collected into a clean vessel, which is placed in a cool place after each urination. All the urine voided in 24 hours is poured into a measuring cylinder.

The passage of an excessive quantity of urine (polyuria) is a characteristic symptom of diabetes mellitus and diabetes insipidus, and of a granular (contracted) kidney; it is also found with resorption of exudate and transudate and administration of diuretics. Complete absence of urinary secretion (anuria) is encountered in certain diseases of the kidneys, corrosive sublimate poisoning, cholera, etc. In certain diseases, especially those of the kidneys, it is highly important to know the quantity of urine passed within 24 hours.

Colour. The colour of the urine depends on the presence of urobilin and other pigments, which lend it a paler or darker hue. The urine is usually straw- or amber-coloured. The colour of the urine varies with the quantity of fluids taken into the body and the presence of various morbid admixtures. In the daytime the urine is watery and its colour is pale. A very pale urine is characteristic of diabetes mellitus and diabetes insipidus. In jaundice the urine is dark, taking the colour of beer. Concentrated urine of a dark colour is seen in patients with fever. After a copious formation of phosphate salts the urine takes a whitish-milky colour. The presence of blood in the urine lends it the colour and consistency of meat slops. Certain medicinal substances change the colour of the urine: santonin to greenish-yellow, methylene blue to dark blue, red streptocid to red.

Transparency. Normally the urine is transparent, but when allowed to stand exposed to the air it becomes cloudy. This cloudiness has no diagnostic value at all. The cloudiness of the normal urine is associated with the formation of salts. Microscopy of the sediment gives the final evidence on the character of cloudiness.

Odour. Fresh urine emits a characteristic odour. When allowed to stand exposed to the air it undergoes decomposition and acquires the stench of ammonia. The ammoniac stench of newly-passed urine points to its decomposition in the urinary passages.

Specific gravity. The specific gravity of urine depends on the concentration of its solid constituents and normally varies from 1.015 to 1.020. It is the larger the higher the level of its solid constituents and the darker its colour; there are exceptions to this rule, however. For example, in diabetes mellitus the urine is very pale; nevertheless, its specific gravity may be as high as 1.040-1.060 due to the presence of sugar. This gravity fluctuates during 24 hours within the range of 1.005 to 1.020, depending on fluid intake which is a sign of adequate adaptation of the kidneys. The specific gravity of urine is estimated by means of a urinometer.

The following two types of urinometers are known: one for measurements to be made within a range of 1.000 to 1.025, the other, from 1.025 to 1.050. The specific gravity of urine is ascertained in a measuring cylinder of 100 ml capacity. When pouring urine into the cylinder the latter is tilted to avoid the formation of froth. The urinometer readings should be taken from the lower meniscus of the urine. The graduation mark which coincides with this meniscus will indicate the specific gravity of urine.

Urine reaction. In the overwhelming majority of cases human urine has an acid reaction, which is mainly due to the common dietary patterns. Persons living on a vegetable diet excrete urine of an alkaline reaction, while those eating mixed or meat foods have an acid urine reaction. The urine reaction is determined by means of red and blue litmus paper. In urine which has an acid reaction red paper retains its colour, while in that with an alkaline reaction it turns blue. Blue litmus paper turns red in acid reactions and retains its colour in an alkaline reaction.

Albumin determination. The presence in the urine of albumin is known as albuminuria. Several tests are used for a quantitative determination of albumin in the urine, but the test commonly used is that with sulphosalicylic acid. A 5 ml quantity of urine is put into a clean test tube, adding 3-4 drops of a 20 per cent sulphosalicylic acid with a pipette. If urine has an alkaline reaction it should be acidulated by adding several drops of acetic acid. In this test urine containing albumin will show cloudiness, the intensity of which will be directly proportional to the amount of albumin in the urine. In order that this cloudiness may be seen, a test tube with urine is examined visually in passing light against a dark background, comparing it with a test tube filled with filtered (control) water. During this test, salts, known as urates, will also be found in the sediment, but if the test tube is heated, this sediment will dissolve, while albumin will remain unchanged. The sulphosalicylic test is highly sensitive. This test may help ascertain an albumin concentration of 0.015 pro mille, which will be seen as a slight cloudiness. Albumin in the urine can also be determined by means of a boiling test. A 10 ml quantity of filtered urine is put into a test tube, acidulated with a few drops of a 3 per cent acetic acid, and boiled. Urine containing albumin will show a white cloudiness, which will not disappear after the urine has been allowed to stand.

The quantitative determination of albumin is based on the reaction of urine albumin to nitric acid. This reaction manifests itself by a white circle appearing after urine has been superimposed on nitric acid. It has been established that if this circle appears in $2^1/_2$-3 minutes, the urine contains 0.033 pro mille of albumin. The larger the quantity of albumin in the urine the earlier the appearance of the circle, while its appearance after the time indicated points to tracer quantities of albumin being present in the urine. In case the circle appears earlier than in $2^1/_2$-3 minutes' time the urine must be diluted until the circle makes its appearance after this time. Urine containing a considerable quantity of albumin is diluted 1 : 200, 1 : 300, 1 : 400. This is performed as follows: a 1 ml quantity of urine is poured into a cylinder and diluted with water to 10 ml, i.e., diluted 1 : 10, whereupon another test for albumin is undertaken. Should in this case the circle appear earlier, the urine is again diluted 1 : 10, 1 : 20, etc., until the circle forms in $2^1/_2$-3 minutes' time. The quantity of albumin is estimated by multiplying 0.033 by the rate of urine dilution.

Example. Urine had to be diluted 1 : 100. Multiplication of 0.033 by 100 gives the figure 3.3, which indicates the quantity of albumin in grams per litre of urine.

Sugar determination. Of practical importance is determination of grape sugar (d-glucose), the presence of which in the urine is called glycosuria. Several tests for a qualitative estimation of sugar in the urine are known. Of them all, Nylander's test for d-glucose is the most accurate.

Nylander's reagent: 2 g of bismuth nitrate is ground in a mortar with 4 g of potassium sodium tartrate (Siegnette salt), dissolved in 100 g of a 10 per cent sodium hydroxide solution and filtered. The reagent is stored in a dark glass vessel. Under these conditions it may remain fit for use for a long period of time. For a qualitative determination of sugar in the urine one puts 5 parts of filtered urine into a test tube and adds 2 parts of Nylander's reagent. The mixture is boiled during 3 minutes. Urine containing sugar will turn black as a result of bismuth nitrate contained in Nylander's reagent being reduced to metallic bismuth in the alkaline medium. During boiling the urine may take a black colour also after a peroral administration of salol (phenyl salicylate), salicylic acid, rhubarb preparations, and antipyrine (phenasone). Therefore, before a test for d-glucose, the patient should not be given these drugs. The sugar content of the urine is determined by means of polarization or fermentation. The polarimetric method consists in that the urine containing sugar turns the plane of polarized light to the right; the readings are taken from a special scale of an instrument called the polarimeter or glycosometer. A polarized ray of light meets on its way an optically active substance which changes the plane of ray rotation by a certain angle, owing to which there is a change in the intensity of light seen in the polarimeter eye-piece. Turning the so-called analyzer in the instrument, uniform illumination of the entire light circle in the eye-piece is effected anew. The number of scale divisions by which the analyzer has to be turned so as to achieve a uniform illumination will indicate the percentage of sugar in the urine. This method is very accurate, but for its effective application the glycosometer test tube should be filled with clear water with no sediment and protected against the entry of air.

Determination of blood. The presence of blood in the urine is determined by means of the guaiac test. Boiled urine, 5 ml, is poured into a test tube and supplemented with a few drops of a newly-prepared alcohol tincture of guaiacum and 10 per cent hydrogen peroxide or turpentine, after which the mixture is shaken. If blood is present, the mixture will turn blue.

Determination of acetone bodies. The urine containing acetone bodies (acetone, aceto-acetic acid, β-oxybutyric acid) emits the odour of rotten fruit. Normal urine contains an insignificant number of acetone bodies; their presence in a large number is a symptom of inadequate carbohydrate metabolism, which is seen in diabetes, fever, etc. Acetone bodies are determined by means of a test using sodium nitroprusside. This solution is prepared immediately before use: 0.5-1.0 ml of water is added into a test tube containing several crystals of sodium nitroprusside and shaken vigorously. The solution turns red, while several crystals of sodium nitroprusside should remain at the bottom of the test tube. A 10 ml quantity of filtered urine is poured into the test tube, 10 drops of glacial acetic acid and 4-5 drops of a sodium nitroprusside solution are added after which the test tube is shaken vigorously, and a stronger ammonia water is superimposed on its contents. If the urine contains acetone bodies, a circle of a violet colour will form on the border between the fluids, its intensity depending on the number of acetone bodies.

Test for urobilin. A 10-15 ml quantity of filtered urine is supplemented with an equal quantity of a 10 per cent alcohol solution of zinc acetate, after which the mixture is shaken and filtered into two test tubes. Into one of them several drops of concentrated hydrochloric acid are added, after which the test tube is examined in side light against a dark background. When the test is positive a green fluorescence is visible. The other test tube is examined in a spectroscope. An increased urobilin content of the urine is encountered in the same pathologic states which are responsible for an increase in urobilinogen.

Bilirubin determination. Bilirubin appears in the urine in the event of an increased level of bile pigments in the blood serum. Urine containing bile pigments forms a yellow froth upon shaking. For an accurate bilirubin estimation an iodine test is employed. A 10 ml quantity of unfiltered urine in a test tube is covered with a 1 ml layer of a 1 per cent alcohol solution of iodine. The green circle appearing on the border between the urine and iodine points to the presence of bilirubin.

For a *nitric acid test* a strong nitric acid with an admixture of nitrous acid is poured into a test tube to one-third of its capacity, after which filtered urine is superimposed upon its wall with a pipette. The presence of bile pigments in the urine is evidenced by the appearance of coloured circles (yellow, green, violet, red, blue) on the border of the fluids.

Determination of indican. Normal urine contains an insignificant quantity of indican. An increased indican content of the urine is found in intensification of the processes of putrefaction in the intestine, decay of malignant newgrowths, suppurative processes and persistent constipation. For determination of indican, filtered urine, 5 ml, is supplemented with 5 ml of concentrated hydrochloric acid, 1 ml of chloroform, and 1-2 drops of a 2 per cent potassium permanganate solution, after which the test tube, preliminarily plugged with a stopper, is shaken several times. In urine containing indican, drops of chloroform will fall to the bottom of the test tube and turn blue.

Examination of the urine sediment. A urine sediment may be obtained by settling or, which is faster, by centrifugation. The latter is performed in special laboratory centrifuges. Centrifugal test tubes are filled with urine to a level 2-3 cm below the rim, set into the opposite nests of the centrifuge and centrifugated for 10 minutes, gradually increasing the rotation speed. Conventional laboratory centrifuges have a speed of 1,000-1,500 rpm. If the quantity of urine is insufficient, another test tube is filled with the same quantity of water for balance. After centrifugation, the fluid part of the urine is poured off quickly, by a single jerk, because slow draining is likely to disturb the sediment. The latter is taken from the test tube bottom with a pipette, placed on a slide and covered with a cover glass, care being taken that the drop is not squeezed out beyond the edges of the cover glass. Microscopy of the urine sediment reveals leucocytes, erythrocytes, epithelial cells—flat, round, tailed (caudate), and renal; casts—hyaline, granular, waxy, epithelial; mucus, and crystalline and amorphous salt sediments. For a general orientation, the sediment is at first examined at a small and then at a large magnification rate. The types of epithelial cells and then leucocytes are counted and determined. All formed elements are counted in one field of vision.

Epithelial cells may be squamous (polyhedral, flattened, large cells), round (deeper large-sized cells of a round or oval form), tailed (deepest pear-shaped cells). The presence in the urine of a large number of epithelial cells is a sign of an inflammatory process in the urinary passages; in normal urine they number 1-2 in the field of vision.

Isolated *leucocytes* occur in the field of vision almost in any normal urine, particularly after a physical exertion. They may be as numerous as to cover the entire field of vision (pyuria), which is evidence of a suppurative process in the urinary passages. In acid urine, the protoplasm of leucocytes is found to have distinct granularity. In order that the leucocytes may be better discernible, a drop of a 3 per cent acetic acid solution is added to the sediment under examination, which eliminates granularity and makes the nuclei distinctly visible.

The permanence of *erythrocytes* in the urine sediment is a sign of a disease. The erythrocytes have a regular form, a darkish colour, and sometimes stick together to form what looks like columns of coins. Erythrocytes having lost their colouring matter are known as achromacytes or phantom corpuscles. Achromacytes are found in kidney diseases. Erythrocytes in the urine are encountered in nephritis, nephrosclerosis, tuberculosis, newgrowths of the kidneys, and cystitis.

The *casts* may be epithelial, granular, hyaline and waxy (Fig. 15). They are formed of the epithelium of the uriniferous tubule, renal epithelium, and coagulated protein in a lesion of the renal parenchyma.

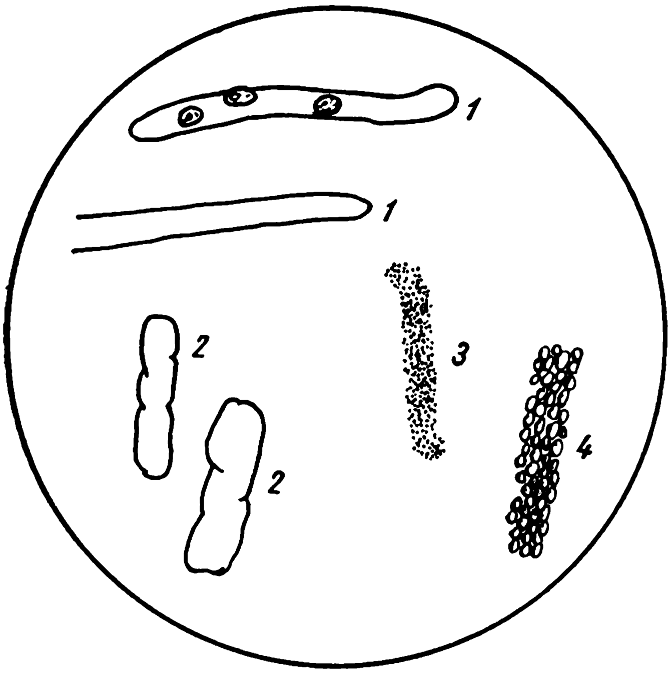


Fig. 15. Casts in urine sediment
1—hyaline casts; 2—waxy casts; 3—granular casts; 4—casts composed of erythrocyte accumulations

Besides the renal casts proper, the urine may also be found to contain casts of a protein origin—blood casts consisting of erythrocytes, casts of leucocytes, and urate casts of salts of uric acid.

Salts occur in the urine sediment owing to a change in the conditions of dissolution, which takes place in certain diseases, as well as when urine is allowed to stand. Precipitates thrown down from acid urine are urates, uric acid, oxalates (calcium oxalate) (Fig. 16), and from alkaline urine—amorphous phosphates, carbonate of lime, triple phosphates, and ammonium urate (Fig. 17). Urates are of a bright-orange, brick-red colour. In microscopy they are seen as numerous granules. A large number of urates are found in patients with fever and in concentrated urine, in a hot weather and in intensified perspiration. Uric acid is seen as yellow-red, variably shaped crystals, which form combinations, the so-called druses. Oxalate deposits form in acid, neutral and alkaline urine, lending the sediment a flaky appearance. Viewed under a microscope, they appear as octahedrons, sharply refracting rays of light. Phosphates (calcium and magnium phosphate salts) form a white or greyish sediment in the urine, shown as fine granular masses by microscopy. They are found in the urine of healthy persons at the time of the most intensive digestion. Triple phosphates are usually found together with amorphous phosphates and in microscopy have a characteristic appearance bearing resemblance to a coffin lid or, less often, fern leaves. Ammonium urate appears in microscopy as crystalline brown globules covered with numerous thorns. Microscopy of calcium carbonates shows colourless granules arranged like gymnastic weights.

Examination of Contents of the Stomach

Collecting gastric juice. Gastric juice is drawn through a thick or thin tube in the morning on an empty stomach and after a test breakfast. A resting stomach usually contains 20-30 ml of fluid and its level above 50 ml is likely to indicate a stomach disease. The thick tube is made of elastic rubber. It is 70 cm in length, 10-12 mm in diameter, and has an 8 mm lumen. The tube end to be inserted into the stomach is rounded and blind; higher up on the sides there are

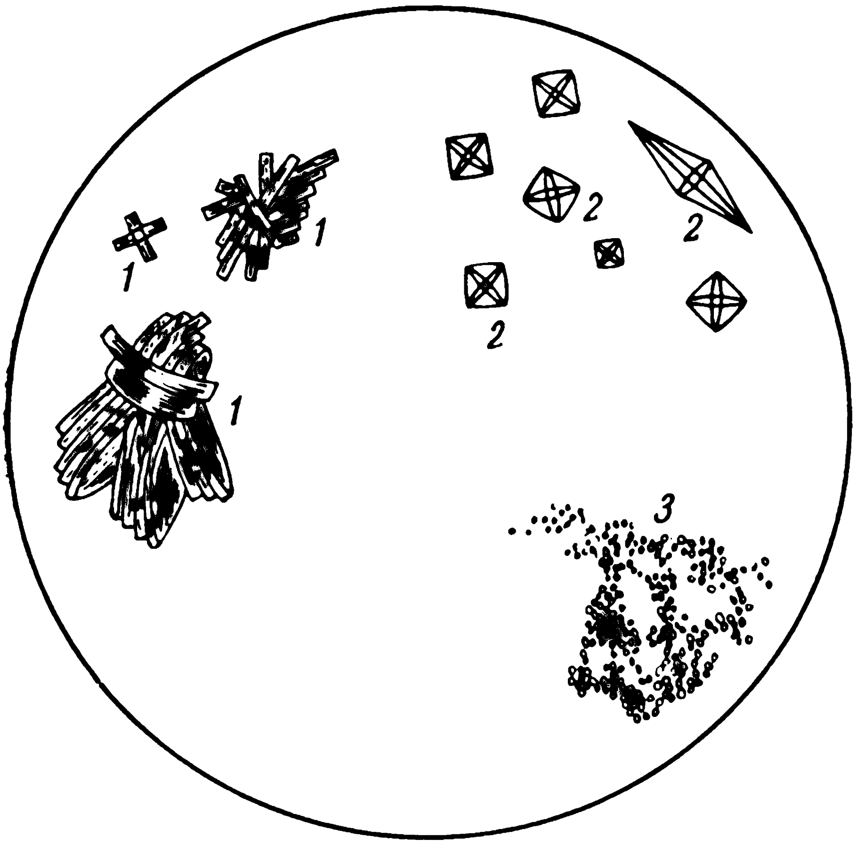


Fig. 16. Crystals in acid urine sediment
1—uric acid; *2*—calcium oxalate; *3*—amorphous urates

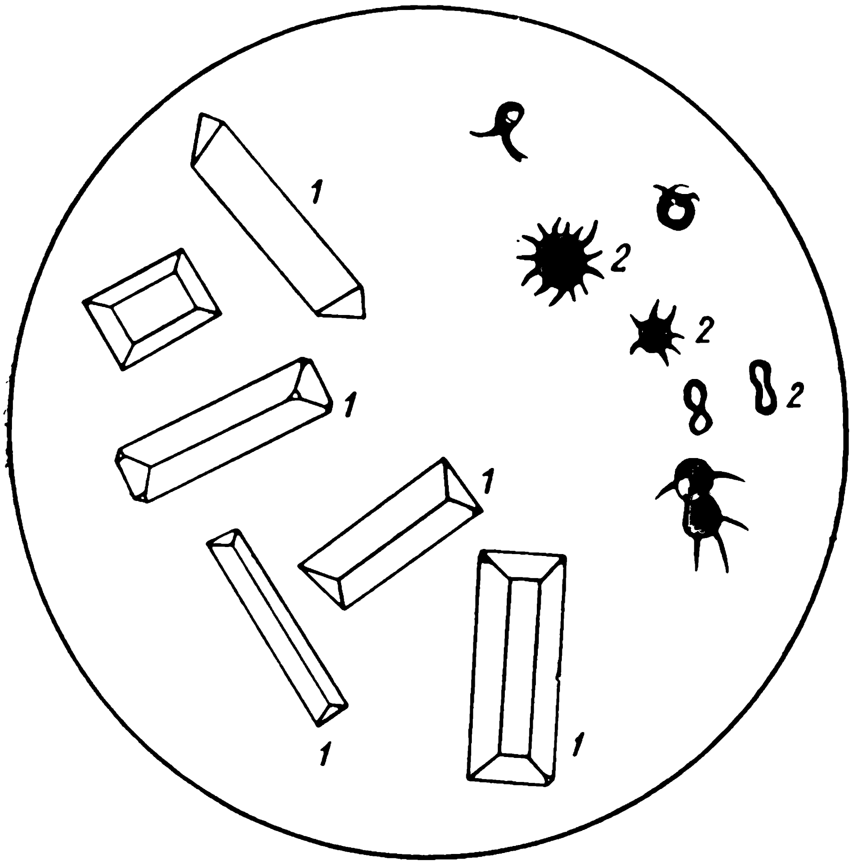


Fig. 17. Crystals in alkaline urine sediment
1—triple phosphates; *2*—ammonium urate

two oval openings through which the stomach contents enter the tube. At a distance of 40 cm from the tube end there is a mark indicating the depth of tube insertion (from the front incisor teeth). The tube is boiled and inserted wet. Before insertion of a tube the patient is told to clear the nasopharynx from mucus; then he is seated on a chair in a slightly bent forward position and helped to put on a rubber apron. The nurse stands to the right of the patient. She holds his neck with her left hand and inserts the tube with her right hand. After one or two efforts at swallowing, the tube easily enters the oesophagus. The patient ought to be warned to breathe through the nose, regularly and deeply, which may be sufficient for suppressing the vomiting reflex. Avoiding jerks, the tube is quickly inserted to the above-mentioned mark, which is usually followed by an immediate flow of stomach contents into a clean vessel kept at hand. Should this fail to happen the patient is told to press the stomach region with his hands. If, however, no gastric juice appears the tube is withdrawn, and the patient is given a test breakfast, inasmuch as a resting stomach sometimes contains no gastric juice.

The most commonly used is Boas-Ewald's test breakfast, which consists of 400 ml of weak tea without sugar and 50 g of dry white bread. The patient is told to chew the bread thoroughly and wash it down with tea or boiled water. The gastric juice is collected into a separate clean flask 45 minutes after the test breakfast. If the bread has been chewed inadequately, bread crumbs may obstruct the tube, and the stomach contents will cease to come up. In this case, the tube should be taken out, cleansed and introduced again. Drawing contents of the stomach through a thick tube sometimes fails to give a complete picture of gastric secretion, therefore, in certain cases a thin tube has to be used.

The thin tube is also made of rubber. It is elastic, measures 4 mm in diameter and 1 m in length, and has a blind end with an oval opening. There is a mark at a 40 cm distance. A thin tube can be inserted only with active help from the patient, who must swallow the probe without haste, breathing through his nostrils. Sometimes, the tube has to be inserted through the nose because of a strong vomiting reflex. When it has been slipped in as deep as the above-mentioned mark (40 cm), the entire stomach contents are pumped out by means of a 20 g syringe. After this, one of the following liquid test breakfasts is introduced into the stomach: (a) caffeine breakfast consisting of 300 ml of water and 0.2 g of pure caffeine (coffeini puri); (b) Leporsky's breakfast of 300 ml of cabbage leaf juice; (c) Ehrmann's alcohol breakfast of 300 ml of a 5 per cent ethyl alcohol solution; (d) 200 ml of meat broth. The investigation is carried out according to a special scheme. There are several such schemes used in medical practice but those of Leporsky and Katch are the most common.

Under Leporsky's scheme, all the contents of a resting stomach are pumped out, after which a test breakfast is given. The first portion is drawn 10 minutes after the breakfast and the entire remainder of the liquid test breakfast in another 15 minutes. Then, the contents of the stomach are drawn into separate test tubes once in every quarter-hour during $1^1/_2$ hours since the moment when the breakfast was introduced. The findings obtained are recorded in a special table.

Under Katch's scheme, the contents of a fasting stomach are drawn once in every 10 minutes during half-an-hour, and then a caffeine breakfast with an addition of several drops of a 1 per cent methylene blue solution is given to determine the stomach motor function. The gastric juice is pumped out every 10 minutes in portions of 10 ml at most until the blue colour has vanished, whereupon the tube is left in the stomach for another 30-60 minutes, depending on the stomach secretion, the pumping being resumed every 10 minutes. The entire procedure should not last longer than 100-110 minutes.

Contraindications for the introduction of a gastric tube are recent stomach haemorrhage, constriction of the oesophagus, aortic aneurysm, heart defects, and a considerably heightened arterial pressure. In such cases, the gastric juice may be investigated without using a tube—by determination of uropepsin in the urine as well as with the aid of ion exchange resin. The presence of uropepsin in the urine is evidence of pepsin and free hydrochloric acid being contained in the stomach contents. After taking ion exchange preparations per os in combination with quinine or azo dyes the latter are detected in the urine if free hydrochloric acid is present in the stomach contents.

General properties of gastric juice. Normal gastric juice is of a yellowish-white, faintly cloudy colour. When bile is present in the stomach contents, the juice turns greenish as a result of conversion of bilirubin into biliverdin, whereas in achylia it retains its yellow colour.

Under normal conditions, the gastric juice emits a sour odour; in the presence of butyric and lactic acids it becomes rancid-sour; in congestion and decomposition of food, putrid.

The gastric juice may contain various admixtures: bile, blood, and mucus from the upper air passages. Signs of diagnostic importance are a uniform pink colour of the gastric juice because of the presence of blood as well as individual streaks of blood, or stomach contents of the colour of coffee grounds. The presence of blood may be due to an injury to the oesophageal and gastric mucosa as well as ulceration of vessels as a result of ulcer, cancer and polyposis of the stomach and sometimes inflammatory processes in its mucous membrane. Stomach contents of the colour of coffee grounds are seen in ulcer and cancer of the stomach, their brown colour being due to conversion of haemoglobin into haematin hydrochlorate under the action of hydrochloric acid.

The mucus in the gastric juice may take its origin from the nasopharynx or stomach. Mucus from the nasopharynx forms separate, sometimes hanging lumps in the upper layer of the gastric juice. Mucus from the stomach is mixed with the gastric juice, and is discoverable owing to its viscid consistency when the juice is poured from one vessel into another.

The presence of a large amount of mucus points to a catarrhal inflammation of the gastric mucosa.

Normally, the gastric juice gives a sharply acid reaction. It is determined by means of litmus paper. Blue litmus paper turns red on contact with gastric juice, which is a sign of an acid reaction. If litmus paper retains its colour, there is reason to suspect complete absence or a low content of free hydrochloric acid.

The acid reaction of gastric juice also depends on the presence of other acids—lactic, butyric, acetic, and volatile fatty acids. Therefore, determination of gastric juice acidity is begun with detection of the presence of free hydrochloric and other acids.

To detect the presence of free hydrochloric acid, a piece of Congo red paper is moistened with the gastric juice under investigation; in the presence of free hydrochloric acid, the paper will turn blue. Besides that, a dimethylamino-azobenzene test is also used. Several drops of filtered gastric juice are supplemented with 1-2 drops of a 0.5 per cent alcohol solution of dimethylamino-azobenzene, which in the presence of free hydrochloric acid changes its colour from orange-yellow to bright-red.

For *lactic acid determination* 5 ml of gastric juice is filtered into a graduated funnel, supplemented with 20 ml of ether and shaken thoroughly; when allowed to settle this mixture forms two layers of which the lower one is discarded, while the upper one is poured into a test tube. A 20 ml quantity of water and 2 drops of a 10 per cent ferric chloride solution are added. In the presence of a 0.05 per cent lactic acid solution the water layer turns greenish, whereas in the presence of a 0.1 per cent solution it takes an intense yellowish-green colour. Fatty volatile acids are determined from their rancid smell.

Quantitative determination of gastric juice acidity. The acidity of the gastric juice (total acidity) is the sum total of free hydrochloric acid, bound hydrochloric acid, otherwise known as protein-bound acid, phosphates and, finally, lactic and acetic acids whose content in normal gastric juice is negligible.

An analysis of gastric juice is carried out within 2-3 hours of its extraction from the stomach, since its acidity changes when it is allowed to stand. Total acidity and free and bound hydrochloric acids are determined. Determined separately are (a) free hydrochloric acid, i.e., that part of hydrochloric acid which is not bound with protein substances, and (b) bound hydrochloric acid, i.e., an acid which forms combinations with protein substances. The acidity is determined by means of titration of a 10 or 5 ml quantity of filtered gastric juice with a decinormal sodium hydroxide solution.

For *determination of total acidity*, into a glass containing 5 ml of gastric juice are added one or two drops of a 1 per cent alcohol solution of phenolphthalein as an indicator and the mixture is titrated with a decinormal sodium hydroxide solution from a burette. Before titration is begun, the level of caustic soda in the burette is accurately measured and registered. Sodium hydroxide is added to the gastric juice until the solution takes a stable, persistent pink colour. Then, the new level of the sodium hydroxide solution in the burette is noted. The amount of sodium hydroxide expended on neutralization of the gastric juice is calculated from this level. The titration is effected against a white background. The total acidity is usually expressed in millilitres of sodium hydroxide solution required for neutralization of 100 ml of gastric juice. Therefore, for estimating the total acidity of the gastric juice, the quantity of decinormal sodium hydroxide solution expended in titration of 5 ml of gastric juice should be multiplied by 20 (or by 10 if 10 ml of gastric juice was titrated).

Example. The level of sodium hydroxide in the burette before titration is 3.6, after titration 6.8; the quantity of solution expended during titration is 3.2 ml (6.8 — 3.6 = 3.2); 3.2 × 20 = 64. Consequently, the total acidity is equal to 64.

For *free hydrochloric acid determination*, the same quantity of gastric juice is put into a glass, adding a 0.5 per cent alcohol solution of dimethylamino-azobenzene as an indicator, which in the presence of free hydrochloric acid turns from yellow to bright red. The titration is continued until the liquid takes a yellow colour. The number of millilitres of decinormal sodium hydroxide expended in titration is multiplied by 10 or 20 to ascertain the quantity of decinormal sodium hydroxide solution required for binding the entire quantity of free hydrochloric acid in 100 ml of gastric juice.

Quantitative determination of total acidity and free hydrochloric acid may be effected in one and the same portion. For this purpose, one drop of dimethylamino-azobenzene is at first added to filtered gastric juice (determination of free hydrochloric acid), whereupon the juice is filtered until it turns yellowish-green rather than frankly yellow. After the quantity of sodium hydroxide expended in titration has been written down, 1 or 2 drops of phenolphthalein are added to the gastric juice (for free hydrochloric acid determination), and titration is continued until the appearance of a stable pink colour, whereupon the new level of sodium hydroxide in the burette is noted.

Example. The level of sodium hydroxide in the burette before titration is 39, by the end of titration with dimethylamino-azobenzene 41, and after the final titration with phenolphthalein 41.8. Consequently, the content of free hydrochloric acid per 100 ml will be (41 — 39) × 20 = 40. The total acidity of the gastric juice will be as follows: (41.8 — 39) × 20 = 56.

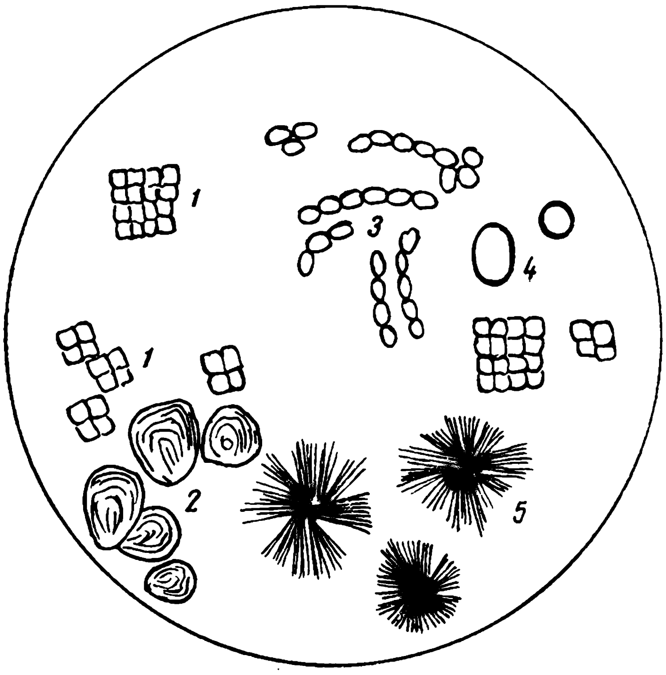


Fig. 18. Microscopy of gastric contents sediment

1—sarcinae; *2*—starch granules; *3*—yeast cells; *4*—fat globules; *5*—needle-shaped crystals of fatty acids

For *determination of bound hydrochloric acid*, a 5 ml quantity of gastric juice is supplemented with 2 or 3 drops of a 1 per cent sodium alizarin sulphonate and titrated with a decinormal sodium hydroxide solution until the gastric juice changes its yellow colour to violet. The calculation is made by means of deduction of the acidity ascertained by titration with alizarin from the total acidity.

Example. The total acidity of the gastric juice is equal to 60; 2.5 ml of sodium hydroxide has been expended on titration of 5 ml of gastric juice with sodium alizarin sulphonate. Consequently, the expenditure per 100 ml of gastric juice will be 2.5 × 20 = 50. The acidity obtained by titration with sodium alizarin sulphonate, i.e., 50 is deducted from the total acidity of the gastric contents, i.e., from 60. The difference 10 will indicate bound hydrochloric acid.

The normal total acidity after Boas-Ewald's test breakfast is equal to 40-60, a decreased one, to less than 40, an increased one, to more than 60. The content of free hydrochloric acid after Boas-Ewald's test breakfast is equal to 20-40. If the quantity of free hydrochloric acid is larger than 40, this is evidence of an increased acidity; if, however, it is smaller than 20, the acidity is decreased.

Detection of blood. In addition to detection of blood in the gastric juice with the naked eye, a benzidine test is undertaken. Unfiltered gastric juice is used for this purpose after its preliminary neutralization with soda.

Microscopy of the stomach contents. For a microscopic examination, a drop of stomach contents taken from the glass bottom is put on a slide and covered with a cover glass. It is at first examined at a small magnification (for a general orientation) and then at a large magnification. The microscopic picture may vary with the functional condition of the stomach. Normally, a resting stomach is found to contain epithelial cells from the oral cavity or cylindrical epithelial cells from the stomach, fresh or phantom red blood corpuscles, leucocyte nuclei (protoplasm being destroyed by the gastric juice), and individual yeast cells (Fig. 18).

An increase in the number of leucocytes points to an inflammatory process in the gastric mucosa. In case of a long retention of the stomach contents microscopy reveals fungal formations, known as sarcinae, looking like bound-up bales. After Boas-Ewald's test breakfast a large quantity of starch is found, better visible upon addition of iodine. When the hydrochloric content of the gastric juice is low, granules of starch may have a laminar structure or completely disintegrate under the action of ptyalin of the saliva. If, however, the gastric juice is found to contain lactic acid, microscopy may show lactobacilli of Boas-Oppler.

Duodenal Drainage

Drainage of the duodenum is very important for identifying diseases of the liver and gallbladder. It is carried out in the morning on an empty stomach by means of a special tube tipped with a small hollow metal olive with holes in its centre. There are marks along the length of the tube, beginning from the olive: the first mark at a 40 cm distance (from the incisor teeth to the cardiac part of the stomach), the second mark at a 70 cm distance (from the incisor teeth to the pylorus) and the third mark at an 80 cm distance (from the incisor teeth to Vater's ampulla). Before its insertion the tube is boiled. The patient is seated slightly tilted forward, the olive is introduced through his widely open mouth, and he is told to make an effort at swallowing. If he is apt to vomit he should be told to take a deep breath through his nostrils, keep calm and continue to swallow the olive. No sooner has the olive reached the stomach than the gastric juice begins to come up. After this, the patient is told to lie on his right side, and a pad is put under his pelvic region, while his legs are bent at the knees and drawn to the stomach. The olive is helped to pass further by gastric peristalsis. It rarely enters the duodenum immediately, and usually it is not before $1\text{-}1^1/_2$ hours later that transparent pale-yellow bile appears which is a sign of the olive being in the duodenum. The yellow colour change in the fluid may also result from the duodenal contents being thrown into the stomach, but its reaction, as distinct from that of the duodenal bile, will be acid rather than alkaline. One making duodenal drainage often has to cope with a number of difficulties. Sometimes, the olive long fails to find its way into the duodenum as a result of the tube twisting within the stomach, which is easily checked by air injection into the tube through a 20 g syringe. During this procedure, the patient will have a clear sensation of air movement in the stomach, while those standing near will hear a characteristic gurgling sound in the epigastrium. In addition, the patient may be given 2-3 mouthfuls of milk, then drawn off with a syringe, which is easy to perform if the probe is not twisted inside the stomach.

The best way to verify this is by x-raying. If the olive has failed to pass through the pylorus, one has to withdraw the tube up to the first mark, and wait again for the olive to pass through the pylorus.

Nevertheless, the olive sometimes fails to enter the duodenum, which is often due to a spasm of the pylorus. In this case, the patient is given an injection of atropine, or if the spasm is due to an increased hyperacidity of the gastric juice, the stomach contents are pumped out, and a small dose of a 2 per cent soda solution is introduced. If duodenal drainage is unsuccessful, it is repeated in 2-3 days' time.

As soon as the supply of clear pale bile from the duodenum (A bile) has ceased, one has to obtain cystic bile (B bile) which is drawn when Oddi's sphincter is open and the gallbladder contracts under the influence of administration of certain cholagogues (bile flow stimulants) such as magnesium sulphate solution, etc. Through a syringe inserted into the duodenal tube 50 ml of a warm 25 per cent magnesium sulphate solution are injected so as to obtain another 30-40 ml of dark-coloured viscid B bile in 15-20 minutes' time. B bile cannot be obtained in obstruction of the cystic duct with a stone (calculus) or during inflammatory processes in the region of the gallbladder cervix. Cessation of the secretion of dark gallbladder bile is followed by the appearance of bile of the same character as that of A bile—liver bile (C bile).

The gallbladder reflex can also be induced by means of a subcutaneous injection of a 1 ml dose of pituitrin solution (5 or 10 units).

Estimation of the results of duodenal drainage. Microscopy of A bile normally reveals up to 5-6 leucocytes in the field of vision. B bile normally may contain up to 15 leucocytes in the field of vision. In inflammation of the gallbladder mucosa, the leucocytes become so numerous as to invade the entire field of vision. Microscopy of cystic bile in cholecystitis due to *Lamblia* organisms shows the presence of these parasites. In order that they may be detected, the test tube containing cystic bile must be wrapped in cottonwool immediately after the bile has been collected so as to prevent cooling, and be sent to a laboratory for microscopy.

In suppurative diseases of the bile ducts and liver microscopy of C bile may reveal a large number of leucocytes.

Examination of Faeces

Examination of faeces may be general and special for determining the presence of blood, eggs of helminths, etc. Faeces in a 50 g quantity are collected into special glass or enamel-coated pots, which should be kept in special cell boxes in a lavatory.

If an investigation is intended to determine blood, three days before collecting material the patient is put on a meat-free diet containing no greens, such as sorrel, spinach, etc. Following an analysis, especially for the presence of ascarides and dysentery and enteric fever organisms, faeces are disinfected even before they are discarded into a sewerage system. For this purpose, the following measures are taken: autoclaving, incineration, and sterilization with boiling water, a 5 per cent carbolic acid solution, a 10 per cent lysol solution or a 50 per cent chloride of lime solution. It takes from 5 to 10 hours to sterilize faeces with the above solutions. Upon mixing faeces with a dry chloride of lime in a 1 : 5 proportion eggs of helminths are killed in 1 hour.

In a person with normal digestion the quantity of faeces discharged in 24 hours is subject to large variations and depends on many factors, including the composition and quantity of ingested food. Upon ingestion of vegetables the amount of faeces is larger, of meat, smaller. A person kept on a mixed diet excretes an average of 100-200 g of faeces in 24 hours.

Normally, faecal discharges are of a round, sausage-like form. Ribbon stool is found with strictures of the anus and tumours of the rectum; the so-called sheep-dung stools occur in colitis with a prevailing spasm. The stool consistence, hard or soft, depends on the presence of cellulose, water, and fat in faecal masses. Liquid faeces occur in flux (diarrhoea), hard faeces in constipation.

The colour of faeces depends on the presence of stercobilin, and is usually brown. This colour changes from the presence of certain substances in food: milk food produces yellow faeces, whereas meat lends a darker hue to faeces than does vegetable food. Prunes, coffee, blood sausages, liver, bilberries, and red grape wines lend faeces a black colour.

In haemorrhages from the upper parts of the alimentary tract the faeces acquire a coal-tar consistency as a result of conversion of haemoglobin into haematin hydrochlorate. In haemorrhages occurring in the lower parts of the intestine, the blood retains its colour. The colour of faeces changes upon administration of certain drugs: in the presence of animal carbon, it turns black, bismuth makes it aspid-grey, iron preparations black-green. Stools of a pea-soup colour and consistency are characteristic of typhoid fever, when the grey-white colour of faeces is due to obstruction of the common bile duct (alcoholic stool).

The stool odour is due to the presence of indole and skatole, and is particularly objectionable upon ingestion of meat, whereas milk food gives almost odourless faeces. In putrefaction processes taking place in the large intestine stools are especially foetid, whereas their sour smell is a sign of a fermentative process.

Faecal discharges may contain the following macroscopic pathological admixtures: helminths or segments of tapeworms, indigestible matter, mucus, pus, blood (light-coloured and discoloured), faecal stones, and bile stones.

Macroscopically, large helminths—ascarides (*Ascaris lumbricoides*)—are discovered the easiest. They are white-pink worms with pointed ends, from 25 to 30 cm in length, the males being shorter and thinner than females and having hooked tails. A thorough examination of children's faeces may sometimes reveal the presence of minute helminths looking like white threads. These are the so-called threadworms (*Enterobius vermicularis*, or *Oxyuris vermicularis* as they were formerly termed).

The segments of the unarmed tapeworm (*Taeniarhynchus saginatus*, formerly termed *Taenia saginata*) are elongated and narrow, and their lumen is clearly seen to have an arborescent (treelike) uterus having from 17 to 35 thin "branches". The segments of the armed tapeworm (*Taenia solium*) are also elongated, but the "branches" of its uterus are wider and fewer (from 7 to 12). The segments of the wide tapeworm *Diphyllobothrium latum* are short and very wide, and its uterus is rosette-shaped.

For expulsion of ascarides it is very important to detect the heads of the above-listed tapeworms in the faeces, because otherwise no treatment may be considered successful. For this purpose, the entire mass of faecal discharge is put into a glass pot and placed on a piece of black paper; then water is added and the mass is thoroughly stirred with a glass stick, after which the floating faecal parts are carefully poured off, and clean water is again added to the sediment. This procedure is continued until a clean, non-floating sediment is obtained. For the last time, the entire contents of the pot are strained through a fine metal or horse-hair sieve. The head is searched for at the thinned end of a worm and examined under a microscope so as to determine the species it belongs to.

Undigested remains of food are found with certain diseases of the gastrointestinal tract and in health (seeds, berry and fruit stones, etc.). Muscle fibres seen as brown threads, remnants of connective tissue shaped as white pieces of thread, and lumps of fat (particularly numerous in flux) are also discovered. Mucus in the form of white flakes or even dense whitish membranes is a sign of inflammation of the mucosa of the small intestine. Pus, often mixed with mucus, is discoverable only if it has escaped from the lower parts of the large intestine, i.e., in dysentery, intestinal tuberculosis, ulcerative colitis, disintegrating tumours of the large intestine, etc.

Biliary, pancreatic, and faecal calculi may be detected after all faecal masses have been collected on a sieve and washed with running tap-water. During this procedure all water should be drained into a separate pail for subsequent disinfection. Faecal masses may be washed in a gauze bag under a stream of tap-water.

The faecal reaction is close to neutral and may be slightly acidic or slightly alkaline, depending on food quality. An intense alkaline reaction is found in putrefactive processes occurring within the intestine; a sharply acid reaction is seen with an intensive fermentative process taking place there. The reaction is ascertained by means of litmus paper which is moistened with distilled water and applied to the faeces. The latter should contain no urine admixture.

Chemical determination of blood in faeces. This procedure is important for determining the presence of haemorrhage, because blood is often undetectable by the conventional visual examination (the so-called concealed haemorrhage). Before examination it is necessary to withdraw meat and vegetables from a patient's diet and protect the gastrointestinal tract against all injuries. The most commonly used are the benzidine test (Gregerson and Boas' test) and the guaiac tincture test (Weber's reaction).

For *a benzidine test*, one is to take 0.02 g of benzidine, 0.08 g of barium peroxide, 4 ml of a 50 per cent acetic acid solution. Once they have been weighed benzidine and barium are kept in dry test tubes. At first acetic acid is poured into a test tube, after which benzidine and barium peroxide are added. In this form, the reagent is fit for use not longer than for half-an-hour. Faeces for investigation are taken with a wooden stick and spread in a thin layer on a slide which is then placed on a piece of white paper. One or two drops of the reagent are applied to the smear and watched for the appearance of a blue colour change. The speed of blue colour change is conventionally marked with crosses. An immediate appearance of a blue colour change is usually marked with four crosses, that occurring in 15 seconds with three crosses, that seen in 1 minute, with two crosses, and that in up to 2 minutes, with one cross. A colour change occurring after two minutes is unimportant, and the reaction is considered negative.

For the sake of verification, the test is repeated several times on samples from one and the same portion of faeces.

For *Weber's test*, it is necessary to have glacial acetic acid, 90 per cent ethyl alcohol, ether, a newly-prepared tincture of guaiacum, ozonized turpentine or newly-prepared hydrogen peroxide, and chloroform. Guaiac tincture is prepared as follows: a small quantity of guaiac gum (taken with the tip of a knife) is put into a test tube, 5 ml of alcohol is added, and the suspension is allowed to stand. Ozonized turpentine is obtained from the common trade turpentine, which is poured into a Petri dish or a plate exposed to light and allowed to stand for 48 hours. It is kept in a plugged dark bottle. The test is performed as follows: faecal masses are thoroughly stirred. Approximately a teaspoonful of the mass is then taken with a clean wooden spatula, transferred into a porcelain mortar and glacial acetic acid is added so as to produce a semiliquid pulp, which is then thoroughly brayed with a pestle and poured off into a test tube. After adding 5 ml of ether to its contents the test tube is tightly stoppered, and rolled on a table during half-an-hour in a horizontal position, then set upright, and the ether layer is drained into a dry clean test tube to which 15-20 drops of guaiac tincture, 30 drops of turpentine or 15 drops of hydrogen peroxide are then added. In the event of a positive reaction, the colour change is blue or light-blue.

Microscopy of faeces. Viewed under a microscope, faeces are found to contain, above all, remains of vegetable food in the form of vegetable fibres, remnants of the cellular skeleton of vegetables and fruit. Connective tissue fibres are discovered in persons with a diminished acidity of the gastric juice. Among these remains one sometimes encounters granules of starch which become better visible upon addition of Lugol's solution. The presence of a large number of starch granules points to a disturbance of the amylolytic function of the gastrointestinal tract. The presence of a considerable number of unchanged muscle fibres retaining their transverse stripes indicates a disorder in the function of the pancreas. A large amount of fat contained in the faeces under examination is a sign of disturbed bile secretion, inadequate function of the pancreas or obstruction of its outlet duct. Fat is seen as minute drops or adipose plaques. For a better determination of fat, the preparation is stained with Sudan (pigment brown). Crystals of fatty acid salts remain colourless, whereas neutral fat and crystals of fatty acids take a bright-orange colour.

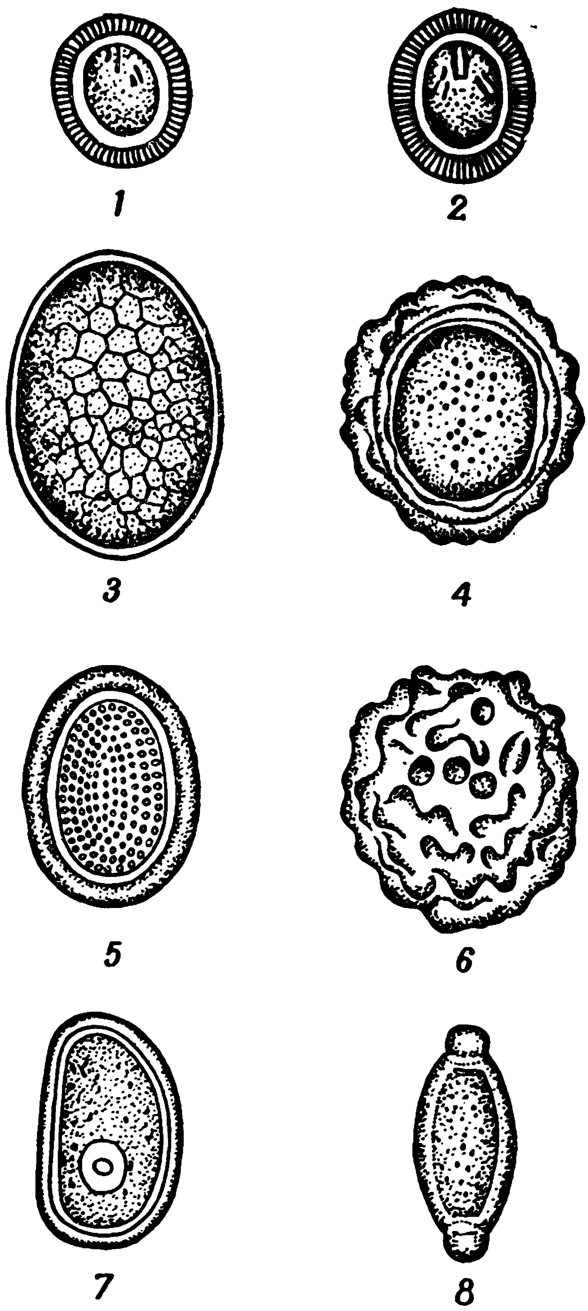


Fig. 19. Eggs of helminths

1—pork tapeworm; *2*—beef tapeworm; *3*—broad tapeworm; *4*, *5*, *6*—ascarides; *7*—pinworm; *8*—whipworm

Examination of faeces for eggs of helminths. Preparation of an ordinary faecal film on a slide does not always answer its purpose. Therefore, a concentrated faecal sediment containing eggs of helminths has to be prepared, for which several methods are used.

The method for preparing a simple sediment. Five or six pieces of faecal matter, each the size of a pea, taken from different parts of the same quantity of faeces are put into a test tube, supplemented with a 15-20 times larger volume of water and thoroughly stirred with a glass stick. The suspension is allowed to stand, after which the liquid part is carefully poured off.

A drop of the sediment is taken with a pipette from the bottom layer onto a slide, covered with a cover glass and examined under a microscope. Several such films must be made.

The flotation (concentration) method. This method is based on the fact that eggs of helminths tend to float in a liquid the specific gravity of which is larger than theirs. This liquid is a saturated common salt solution (400 g per litre of water). Several pieces of faecal matter are mixed with this solution in low glass vessels or a test tube of 15-16 mm in diameter. The common salt solution is added to the mixture up to the upper edge of the test tube so as to form a convexity on which a cover glass is placed. An hour later, the cover glass is carefully removed and placed on a slide, onto which the eggs of helminths which have stuck to the cover glass are thereby transferred. If appropriate vessels or test tubes are unavailable the mixture may be prepared in a conventional porcelain dish. After an hour, the drop is removed from the surface with a piece of thin wire and transferred onto a slide.

The scrape method. This method is used for detecting threadworm eggs deposited by female parasites in the folds of the anus in the terminal portion of the rectal mucosa. Matter is scraped from the above-mentioned parts with an obliquely cut wooden rod or a match moistened with 50 per cent glycerol and further is removed from the rod with the edge of a slide into a drop of glycerol on another slide and examined under a microscope (Fig. 19).

The unarmed tapeworm, also known as beef tapeworm, more often leaves its larvae—oncospheres—in faecal masses. The oncosphere is 23 to 30 mm in size and has a round form. Six rudimentary hooks may be discovered within its dark, radially striped envelope.

The armed tapeworm, otherwise called pork tapeworm, is distinguished from the above-described unarmed tapeworm with difficulty. Therefore, after discovering an egg or oncosphere one usually only indicates that it belongs to the family of tapeworms.

Mature eggs of the broad tapeworm are usually large and oval in form, of a dark-brown colour, with a double-contour envelope, and granular contents. They are 55 to 75 microns in size.

The ascaris lays large oval eggs of dark-brown colour, with a thick festoon envelope. It is 50 to 70 microns in size. An unfertile egg has a knobby surface and measures 80 to 90 microns.

Pinworm eggs are asymmetrical, with a light-coloured, double-contour envelope, containing a larva of about 50 microns in size.

Diseases of the Respiratory Tract

AN OUTLINE OF ANATOMY AND PHYSIOLOGY

The respiratory organs form a complex system, which includes the upper air passages (the nose with its accessory, otherwise called paranasal, sinuses, and the larynx), the trachea, the bronchi, the lungs, the pleura and the diaphragm. The entire thorax with its respiratory muscles and the nerve apparatus takes part in the breathing act. The thoracic skeleton consists of the thoracic part of the vertebral column, the ribs, and the sternum. The upper opening in the thorax is formed by the 1st thoracic vertebra, the 1st pair of ribs, and the manubrium of the sternum. The lower opening is formed by the 12th thoracic vertebra, the 11th and 12th ribs and the xiphoid.

The lungs are a paired organ. The right lung consists of three lobes: upper, middle, and lower; the left, of two lobes: upper and lower, the upper lobe containing a "lingular" division corresponding to the middle lobe of the right lung.

The lungs are cone-shaped and have the mediastinum lying between them. The bronchial branches and blood vessels enter the lungs through the lung hilum, and the latter together with its lymphatic glands forms the root of the lung. The lungs, the interlobar spaces, the root of the lungs, the ental surface of the thorax are lined by a moist serous membrane called the pleura.

In normal respiration, atmospheric air enters the lungs in the following way: through the nose and its meatuses (passages) into the choanae (posterior nasal orifices), the pharynx, the narrow cleft of the larynx (rima glottidis), and the trachea. On a level with the 5th thoracic vertebra the trachea forks into two large bronchi, which enter the right and the left lungs. These large bronchial trunks successively ramify into bronchi ranging in calibre from medium to small and further subdivided into bronchioles which enter the alveoli, minute air cells having division walls. The alveoli are enveloped with capillary vessels on the outside and lined with a squamous epithelium on the inside. A group of alveoli connected with one of the terminal bronchi make up a pulmonary lobule, which is shaped as a pyramid, its wide foundation facing the lung surface and the apex directed inwards.

Even before its delivery to the alveoli, atmospheric air is cleaned of dust particles and bacteria, humidified and warmed upon contact with the moist and warm mucous membrane lining the air passages. Microbes are also captured here. A highly important role in the destruction of bacteria is played by the tonsils and fauces which are profusely supplied with lymphoid tissue, as well as the trachea and bronchi whose mucous membrane is coated with a ciliated epithelium.

The lungs participate in the general circulation as its smaller part called the pulmonary circulation. The latter begins in the pulmonary artery issuing from the right ventricle of the heart and ramifying consecutively into a system of minute capillaries which further communicate with venous capillaries and pulmonary veins draining into the left atrium, where the pulmonary circulation is completed. It is on this route, specifically in the arterial capillaries, that the interchange of blood gases takes place in which carbon dioxide is given off and oxygen is taken up.

The lungs proper and the bronchial tree are supplied with blood through the bronchial arteries branching off from the vascular system of the general circulation. The pulmonary arteries partly anastomose (communicate) with the bronchial arteries, owing to which a certain quantity of blood is drained from the pulmonary circulation vessels into those of the general circulation if there is blood congestion (stasis) in the pulmonary circulation.

The act of breathing consists of inspiration and expiration. During inspiration, the thorax expands in the anteroposterior and lateral directions. This expansion is also due to contraction of the diaphragm, which comes down and displaces the liver, stomach, intestines and spleen downwards. Inspiration is effected by an active contraction of the respiratory muscles, in expiration the thorax is collapsed, and the diaphragm elevated. In inspiration, the thoracic circumference is increased, in expiration it is decreased. During respiration there is an interchange of gases between the venous blood delivered through the pulmonary artery and the atmospheric air. The oxygen taken up from the air by the blood converts haemoglobin of the erythrocytes into oxyhaemoglobin, while carbon dioxide given off by the blood is released into the atmospheric air during expiration. Owing to an interchange of gases the blood is oxygenated, which is indispensable for normal tissue respiration. An adult person performs 16-20 respirations per minute. During physical exertion and excitement, the respiration is accelerated. At rest and in sleep the number of respiratory movements is reduced.

Very important for normal respiratory processes is not only the number of respirations per minute but also the volume of the air inhaled and exhaled, which is determined by spirometry.

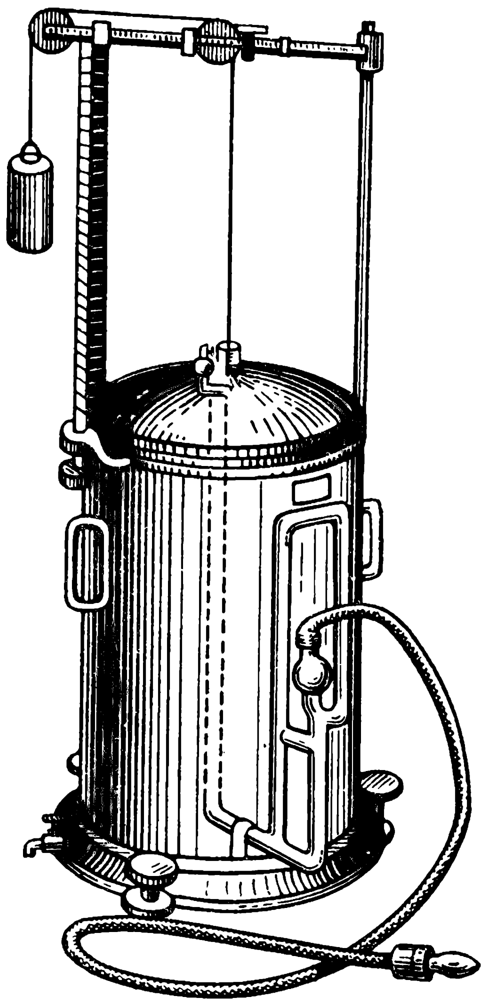


Fig. 20. Spirometer

The spirometer (Fig. 20) is an apparatus consisting of two cylinders, one mounted within the other, the inner cylinder being provided with a rubber tube and a mouth-piece. Under pressure from the air blown into the mouth-piece the inner cylinder rises to a height corresponding to the volume of this air. The volume of air expelled is read from a scale on the wall of the inner cylinder. Normally, the quantity of the air inhaled and exhaled ranges from 500 to 800 cc (tidal air); the volume that can be taken in additionally by a forcible inspiration ranges from 1,500 to 1,600 cc (complemental air), and the same quantity of air can be expelled from the lungs by a forcible expiration following an ordinary expiration (reserve air). After the expulsion of reserve air the lungs will still retain 500 cc of air (residual air). For normal redox processes in the body, very important is the vital capacity of the lungs which is the sum total of the tidal air, complemental air, and reserve air. In men it is equal to 3,000-4,000 cc, in women to 2,000-3,000 cc. In certain pulmonary diseases—tuberculosis, pleurisy, pneumonia, and emphysema, as well as in pneumothorax, the vital capacity of the lungs is considerably diminished, which is responsible for a derangement of normal tissue respiration.

The act of breathing is performed automatically, and is controlled by the centre of respiration in the medulla oblongata. Thermal stimuli acting upon the centre of respiration, as well as certain chemical substances, for example, carbon dioxide and various organic acids accumulating in the body during such diseases as diabetes and uraemia, cause an increase in the depth and number of respirations per minute. A disturbance of the respiratory rhythm is seen with disorders in the blood supply to the centre of respiration (sclerosis of cerebral vessels, cerebral haemorrhage).

SYMPTOMS OF DISEASES OF THE RESPIRATORY TRACT

Acute and chronic pulmonary diseases are due to a variety of causes —airborne droplet infections (measles, whooping cough, influenza, bronchitis), occupational hazards (dust, chemical substances, etc.), unhealthful housing and living conditions, chilling, an irregular diet, physical and mental fatigue, alcoholism, etc. Therefore, in history-taking it is necessary to find out about the patient's conditions of life, occupational surroundings, diseases sustained in the past, cases of transient fever due to unknown causes, whether there is emaciation, a reduction or total loss of the appetite for food, periodic haemoptysis (blood-spitting), expectoration of sputum and its character, and a past record of recurrent pneumonia. Pulmonary diseases sometimes result from a chronic cardiovascular insufficiency, trauma, thoracic injury, etc.

Coughing (tussis) is an important symptom of a lung and bronchial disease. The act of coughing is preceded by a deep inspiration followed by a convulsive expiration caused by contraction of the abdominal and bronchial muscles. In the beginning of a cough the glottis (rima glottidis) is closed, but the increase in the intrathoracic pressure due to this closure causes the glottis to open and expels along with the air the sputum containing various admixtures (blood, mucus). Coughing is caused by a reflex stimulation of the coughing centre located in the medulla oblongata near the centre of respiration and by irritation of the mucosa of the air passages with sputum and other morbid matters present there.

The intensity, duration, and timbre of a cough depend on the character of a disease. Dry cough is consequent upon a sharp swelling of the mucous membranes of the air passages in the presence of a scanty secretion, and is found in dry pleurisy, bronchitis, and the initial stages of lobar pneumonia. A "barking" cough is very typical of a laryngeal lesion, a resounding cough—of a disease process in the lungs, more often a suppurative one, forming cavities. Pulmonary emphysema is often attended with a muffled cough.

Coughing expels secretion from the air passages, therefore its suppression is not always useful. In case there is blood, mucus, and foreign bodies in the air passages, one is advised to remove them and prevent atelectasis of the lung and pneumonia by increasing the excitability of the centre of coughing by artificial means. A painful dry cough ought to be eliminated.

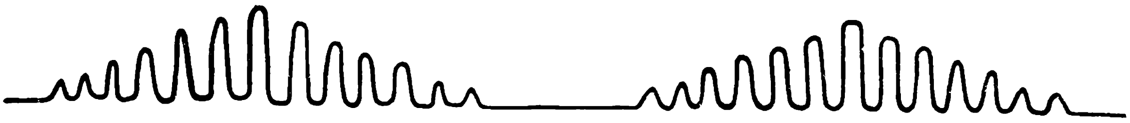
Dyspnoea. This is an accelerated, difficult respiration with a sensation of air deficit.

Dyspnoea is encountered in various affections of the lungs, cardiovascular system and brain, and in healthy persons after physical overexertion, apparently, as a result of a heightened oxygen consumption and an excessive accumulation of carbon dioxide in the blood. Dyspnoea is produced by a reflex stimulation of the brain cortex, as is evidenced by its appearance in agitation and emotional unrest.

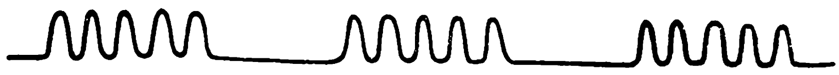
Dyspnoea may be of the following types: (a) inspiratory; (b) expiratory; and (c) of a mixed type. Inspiratory dyspnoea is due to obstruction of the larger air passages by a foreign body, diphtheritic membranes, a polyp and other tumours, or their compression with a goitre. In this type of dyspnoea, the air is inhaled with great difficulty, passing through the narrowed part, with a loud noise, sometimes with a characteristic whistling sound (stridulous respiration) heard from a distance. In inspiratory dyspnoea the respiratory muscles are strained, and the intercostal spaces are drawn in. Expiratory dyspnoea is seen with a considerably difficult exhalation, which occurs in persons with a reduced elasticity of the pulmonary tissue, or constriction of the lumina of smaller bronchi (pulmonary emphysema, bronchial asthma). The auxiliary respiratory muscles also take part in the act of expiration. The third, mixed type of dyspnoea is the most frequently seen. In this condition, both inspiration and expiration are difficult to perform. It is encountered in heart disease, diminution of the respiratory surface of the lungs due to an extensive infiltrative process (tuberculosis, pneumonia), as well as in emphysema and pneumosclerosis. An intense dyspnoea is found with spontaneous pneumothorax, total pneumonia and exudative pleurisy.

Asthma is a severe dyspnoea suddenly coming on as an attack. It is seen in lung and heart diseases. Heart patients suffering from dyspnoea prefer a sitting or half-sitting posture (orthopnoea), whereas lung patients, even with a severe dyspnoea, may find relief in the horizontal position also.

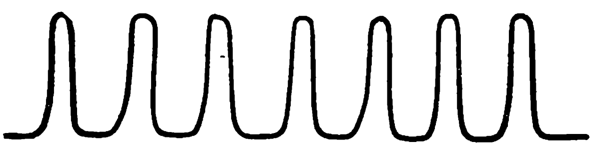
It is necessary to emphasize the importance of a disorder in the respiratory rhythm. Normally, the respiration in adult persons is rhythmical with regular pauses between inspirations. In certain diseases of the brain, heart, and kidneys and in intoxications, however, the respiration becomes arrhythmic. In some cases, prolonged pauses appear, sometimes lasting half-a-minute, after which the respiration is gradually intensified, and the respiratory movements become deeper; after a few deep breaths, however, the respiratory movements become increasingly shallow and finally cease altogether



Cheyne-Stokes respiration



Biot's respiration



Kussmaul's respiration

Fig. 21. Types of respiration

for a certain period. This type of respiration is known as periodic or Cheyne-Stokes respiration (Fig. 21). There is also Biot's respiration.

In diabetes mellitus, uraemia and other diseases, one may meet with what is known as extreme or Kussmaul's respiration, which essentially consists in a noisy breathing, in which prolonged and deep inspiration is followed by a brief expiration and a long pause. Kussmaul's respiration (Fig. 21) is due to severe irritation of the centre of respiration by an abnormal accumulation of acids (acidosis).

Expectoration of blood (haemoptysis). Expectoration of blood is a frequent sign of a lung disease attended with destruction of the pulmonary tissue and rupture of pulmonary vessels and bronchi or rupture of vessels of the adjacent organs into the bronchi (oesophageal cancer, aortic aneurysm). One ought to discriminate between bleeding and expectoration of blood. The former is effusion of a large quantity of bright-red blood, which is more often seen after a rupture of larger pulmonary vessel. In haemoptysis the blood is found in sputum as an admixture or expectorated as separate spits. Both conditions are encountered in tuberculosis, newgrowths, suppurative processes, thoracic injuries, as well as in mitral stenosis, pulmonary infarction, etc.

Pain. Pain in the thorax occurs in persons with chest injuries, pleurisy, newgrowths, pneumothorax, intercostal neuralgia, myositis, etc. Excruciating pains of sudden onset most commonly occur at the beginning of spontaneous pneumothorax. In pleurisy pain in the chest is at first confined to a small area, becoming more intense in a deep inspiration. Newgrowths produce a constant and dull pain. Pains due to neuralgia of the intercostal nerves extend along the ourse of the latter.

EXAMINATION OF THE RESPIRATORY ORGANS

Examination of the Chest

The examination is begun with an inspection of the chest. For this purpose, the patient should best be seated or told to stand in front of the examiner, a side light being used for his inspection. Attention should be paid to the colour of the chest skin (blueness, lividness). Persons with mediastinal tumours have dilated skin vessels on the chest as a result of collateral circulation consequent upon compression of larger vessels by a tumour or enlarged lymphatic glands. During inspection, one takes note of the characteristics of the thorax, indicating its structural type, symmetry of its halves and abnormalities in the excursion of various parts of the thorax.

The thorax along with the muscles and shoulder girdle forms a cone, its foundation facing upwards. The thoracic skeleton proper, without the muscles and shoulder girdle, forms a cone whose foundation faces downwards. The thoracic skeleton consists of the vertebrae, ribs, sternum and clavicles. In adulthood, the vertebral column has the following four curvatures: forward prominence or convexity in the cervical and lumbar part; backward prominence or convexity in the thoracic and sacral regions. The first six ribs are directly connected to the sternum with a cartilage, while the 7th, 8th, 9th and 10th ribs converge on the sternum and are interconnected by means of ligaments. They form the epigastric angle which normally is equal to 90°. The 11th and 12th ribs have no ventral attachment (floating ribs). On the inside of the thorax, on a level with the xiphoid is attached the diaphragm, which separates the organs of the thoracic cavity from those of the abdominal cavity. The shoulder-blades lie between the 2nd and 7th ribs and, when the arms are lowered, closely adjoin the trunk. The sternovertebral dimension is somewhat less than the transverse one. Apart from the thoracic form described one is likely to meet with a number of its variations.

The paralytic or asthenic thorax. A paralytic thorax is flat, narrow, and long. The sternovertebral and transverse dimensions are diminished,the intercostal spaces are wide, the epigastric angle is acute, the supra- and intraclavicular fossae are indrawn, the shoulder-blades are detached from the trunk, the muscles are underdeveloped, and a floating 10th rib is encountered sometimes. A paralytic thorax is found in persons with a bilateral cicatricial pulmonary tuberculosis and contractive processes in the pleura, which lead to shrinkage of the lungs and reduction of their volume.

The emphysematous thorax. The emphysematous thorax is the direct opposite of the paralytic one. It is short, with enlargement of all dimensions, and resembles a barrel in shape. The supra- and infraclavicular spaces are bulging, the intercostal spaces are narrowed, the ribs are horizontal, the epigastric angle is larger than 90°, the relation between the anteroposterior and transverse dimensions is considerably above normal. This form of the thorax is encountered in acute and chronic distention of the lungs.

Rachitic or chicken thorax (pigeon breast). A thorax of this form has indrawn sides, a sharp forward protrusion of the sternum, and rosary-shaped knobs at the junctions of the rib cartilage and bones. A rachitic thorax forms by reason of a disturbed skeleton growth in rickets.

The funnel-chest. In a thorax of this form, a funnel-shaped depression is seen in the region of the lower sternum and xiphoid, which is a developmental anomaly and has no practical significance.

Asymmetry of the thorax. Normally, both halves of the thorax are symmetrical and take part in the act of respiration simultaneously. Upon accumulation in the pleural cavity of a considerable quantity of liquid or air, and in patients with tumours in the lungs or chest wall, one or the other half of the thorax will correspondingly bulge and lag behind during respiration. A retraction of one half of the thorax is seen in contraction of the lungs and pleura owing to inflammatory processes.

The respiratory movements. Under normal conditions, the respiratory movements are performed simultaneously in both halves of the thorax. A bilateral reduction of the lungs excursion is encountered in emphysema, a unilateral one—in exudative pleurisy, pneumothorax, adhesions of the thorax to the pleura, atelectasis of the lungs, etc.

During inspection, one ought to find out the frequency of respiration and the correlation between the latter and the pulse rate which normally is equal to 1 : 4. The rate of respirations should be estimated imperceptibly for the patient because the latter, when aware of being observed, will unwittingly begin to take shallow and rapid breaths. In examination of the respiration its rhythm and depth are also determined. For its type the respiration may be costo-inferior, otherwise known as male (since it is seen mainly in men) and costo-superior, otherwise called female. A knowledge of the type of respiration is important in view of the fact that in some diseases of the lungs and pleura the type of respiration is subject to change. For example, thoracic tumours provoke costo-inferior respiration, whereas diseases of the abdominal cavity, particularly acute abdomen, are likely to cause costo-superior respiration.

Examination of the spine and fingers. Inspection may help detect a number of acquired or congenital malformations in the vertebral column. A curvature of the spine with a forward convexity is called lordosis, with a convexity turned dorsad—kyphosis, and with a lateral convexity—scoliosis. These curvatures sometimes occur in combination. Of these the most common is kyphoscoliosis, i.e., a curvature turned dorsad and laterally. A spinal deformity is usually a sequel of rickets or tuberculosis, and leads to a change in the position of the lungs and mediastinal organs.

In identifying lung diseases, examination of the fingers is also important since clubbed fingers are a characteristic sign of bronchiectasis and abscesses. In such cases, the nails have a curvature over the finger-ends. These changes in the shape of fingers and nails may also occur in some other diseases (subacute bacterial endocarditis, etc.).

Palpation of the Chest

Palpation of the chest is performed by a direct compression of the thorax with the hands as well as by a gentle manipulation of a part with half-bent fingers. The sensations thus received may give an idea of the resilience of the chest walls. When the pleural cavity is filled with fluid or there is a large tumour or infiltration present in the lungs the thorax offers considerable resistance to pressure applied to it. Palpation also helps detect a tender muscle tension often present over an inflamed part of the lungs or pleura. Superficial tenderness is found in inflammation of the soft tissues and in lesions of the intercostal muscles, ribs and sternum. Rib fractures produce crepitation under finger pressure. Painful points along the intercostal space are characteristic of intercostal neuralgia.

Palpation is also used for examination of the vocal vibrations in the chest (fremitus pectoralis, vocal fremitus). In healthy persons a palm applied to the symmetrical places of the chest feels vibrations caused by speaking, particularly in words with many vowels and the sound *r* (ringing, bringing, lingering, etc.). The vocal vibrations may be intensified or diminished. Important diagnostically is a unilateral increase or decrease of vocal vibrations. The processes improving the sound conduction of tissues tend to intensify vocal vibrations, for example, lobar pneumonia in the stage of hepatization. And conversely, in processes which are likely to diminish sound conduction, vocal vibrations are weaker (in accumulation of fluid in cavities, blockage of the adductor bronchi, etc.).

Percussion of the Lungs

Percussion as a method of examination was introduced into medical practice by Auenbrugger in 1762. This method is based on physical factors by reason of which different substances when struck gently give out vibrations of a variable frequency and range, which are perceived as sounds by the human ear. The number of vibrations per second depends on the resilience and elasticity of a given substance, in our case, bodily tissues. Frequent vibrations produce a high-tone sound, slow ones are heard as deep sounds. The volume of sound is determined by the range or amplitude of sound waves. Percussion of organs and parts consisting of dense tissues and fluids elicits a dull or low sound resembling that heard in percussion of the thigh (femoral dullness). Percussion of organs containing no air—the spleen, liver, etc.—also elicits a dull resonance, though not femoral, because air vibrations in the lungs and the abdominal cavity are brought into play. In percussion of the organs of the thoracic cavity the character of sounds depends on the amount of air contained in them, as well as on the closeness of an air cavity to the part or region percussed, since air is highly resilient and capable of sharp vibrations. The soft or dense parts of the human body give few vibrations by themselves but they effectively conduct vibrations of the air they contain.

Percussion may be immediate and mediate.

Immediate percussion. Immediate percussion is performed by tapping a part with the flesh of the finger. V. P. Obraztsov suggested a method of percussion with the index finger, the edge of which is detained on the middle finger and then allowed to slip and tap the part under examination (flick method). Immediate percussion may also be performed with three half-bent fingers pressed to each other.

This method is less convenient because on the one hand, it fails to detect changes in the local percussion sounds since the procedure involves a considerable area and on the other hand, makes it difficult to strike a part with a force sufficient to elicit sounds from the underlying parts. An advantage of immediate percussion is the possibility to use the sense of touch, which helps determine the degree of resistance offered by the underlying tissues.

Mediate percussion. This is percussion with finger strokes at another finger applied to the body. A bent third finger of the right hand strikes the third finger of the left hand at the second phalanx firmly, though without great effort, pressed to the part under examination. Finger strokes should be perpendicular to the interposed finger. A stroke must be quick and brief, and applied by a free motion of the hand.

Percussion may be loud, or deep, and quiet, or superficial. In a loud percussion the vibrations of the tissues examined extend up to 6-8 cm deep and 4-6 cm wide. Quiet percussion reaches a depth of 4-5 cm and a width of 2-3 cm. It is employed in case the area under examination lies in close proximity to air-filled cavities, also when it is small and lies near the surface. The use of loud percussion in such cases evokes vibrations of the air in the adjacent organs, so its findings fail to reflect the condition of the part under study. This type of percussion is used when an organ or region to be examined is located at a considerable depth.

For mediate percussion a special hammer called a plexor and a pleximeter (from Greek *plexis*—stroke and *metron*—measure) may be used instead of a finger. In such a case, the latter is firmly pressed to a part and struck with a plexor. If a plexor is unavailable the pleximeter may be struck with the rib of a coin or with a metal rod. This method is used mainly for percussion of air-filled cavities. The sounds thus elicited have a metallic tone.

Percussion sounds may be loud, or clear, and soft, or dull, high-pitched and low-pitched, and tympanitic and nontympanitic in resonance.

A loud and a soft percussion note (clear and dull) vary in proportion to the range of sound vibrations. The range depends on the force of a stroke, the resilience of the part percussed, and the presence of air in the percussion region. Percussion strokes ought to be of equal force, because strong strokes elicit loud sounds and weak strokes soft ones. Under normal conditions, the force of strokes being equal, a clear sound is heard on percussion of the chest in a region with an air-filled cavity. A soft sound is produced on percussion of air-free organs and soft tissues (heart, liver, spleen, muscles, etc.). In addition to clear and dull sounds, there is also an intermediate, muffled or deadened, percussion note. A dull or muffled sound instead of a clear one is heard when the amount of air in the part examined is reduced in volume or absent altogether. This takes place in patients with lobar pneumonia, newgrowths, infarction and pulmonary atelectasis. Dull percussion notes over the lungs are also heard in exudative pleurisy owing to fluid accumulating between the chest wall and the lungs as well as in persons with a thickened pleura in consequence of fibrinous deposits reducing the air conduction of tissues.

A sound is high or low in pitch, depending on the frequency of vibrations in a vibrating body. The greater the vibration frequency, the higher the sound pitch. The latter is always connected with other characteristics of sound. A high-pitched sound is at the same time short and soft, while a low-pitched sound is loud and long. Sound quality also depends on the volume of the vibrating body: the less the volume of the body, the higher and shorter the sound, and conversely, the larger the volume, the lower and louder the sound. In pulmonary emphysema the sound is low-pitched, loud, and is known as a bandbox sound because it seems to have been produced by striking an empty box.

The tympanitic percussion note is close to the bandbox sound and is like that produced by a tympanum (drum), from which it takes its name. This sound is elicited on percussing smooth-walled hollow air-distended organs. Normally, tympanitic resonance is obtained on percussion over the oral cavity, the larynx, the trachea, the stomach and the intestine. In the lungs it is heard in the presence of cavities, an open pneumothorax, as well as at the onset of lobar pneumonia and other infiltrative processes when the lung tissue tension is reduced. The tympanitic percussion note takes a metallic tone on percussion above large caverns and open pneumothorax.

Topographic percussion. This type of percussion is undertaken with a view to demarcating the organs under examination from the adjacent organs, as well as for determining their shape and size. It is very difficult to perform when the boundaries of adjacent organs with a variable air content and overlapping one another have to be determined. For example, under certain conditions some parts of the heart are partly covered by the lungs. In such cases, one is likely to meet with certain difficulties in determination of the heart borders, since the sounds elicited on percussing the parts covered by the lungs will differ from those coming from the exposed parts of the heart.

Topographic percussion is highly important for determination of the lung boundaries. This procedure is based on the fact that the normal lungs upon percussion give out a loud (prolonged) note, whereas the organs adjacent to the lungs, except for the stomach and intestine, give out a dull percussion note.

The lung boundaries are determined according to plan. At first, one determines the borders of the lung (apices), which is the most difficult, since they are partly covered by a thick layer of muscles and by bones. Anteriorly, the upper boundary of the lungs lies 3-4 cm above the clavicle (Fig. 22), and posteriorly on a level with the 6th-7th cervical vertebrae (Fig. 23). The lung apices are determined by a soft percussion carried from below upwards, i.e., from a clear to a dull resonance tone since a loud percussion, extending to the deep-lying regions of the lungs, may give a distorted picture of their true boundaries. The lower boundaries are also determined by soft percussion. For this purpose, a finger of the left hand or pleximeter is placed parallel to the boundary searched for and along the ribs. At first, one has to determine the lower boundary of the right lung and then of the left lung, for which the intercostal spaces are percussed from above downwards along the following five lines: parasternal, midclavicular, axillary, scapular, and paravertebral. The lower boundaries of the lungs on both sides are indicated by the lines of conversion of a clear pulmonary into a dull sound. Normally, the boundaries of the right lung lie along the parasternal and midclavicular lines on the 6th rib (the parasternal boundary of the left lung is the 4th rib), along the anterior axillary line—on the lower margin of the 7th rib, along the midaxillary line—on the 8th rib, along the posterior axillary line—on the upper margin of the 9th rib, along the scapular line—the 10th rib, and the paravertebral line—at the spinous process of the 11th thoracic vertebra. The lower boundary of the lungs is a horizontal line with a small notch only in a limited section where the heart immediately adjoins the thoracic wall.

The normal lung boundaries are subject to change, depending on the physiologic and pathologic factors. The lower boundary is raised in pregnancy, in persons with a hypersthenic constitution, and is depressed in persons with an asthenic constitution.

More often a change in the boundaries is found in pathologic states of the body. For example, a depression of the lower boundary occurs in pulmonary emphysema, and in severe prolapse of the viscera and diaphragm. Elevation of the lower boundaries of the lungs is seen in diseases of the lungs and adjacent organs. A unilateral elevation of the lower boundaries takes place in all contraction processes in the lungs and the pleura. Of the extrapulmonary processes causing a bilateral elevation of the lower boundaries, the most important is heightened intra-abdominal pressure as a result of fluid accumulating in the abdomen, of meteorism, newgrowths, an enlargement of the liver or spleen, etc.

Determination of the active mobility of the lung margins. The active mobility of the lung is ascertained by finding the difference between the lower boundaries at maximum inspiration and at maximum expiration. For this purpose, the first step is to determine the lower boundary during an ordinary respiration, after which the patient is told to draw a deep breath and hold it at its maximum. After these boundaries have been determined the patient is again called upon to expel as much air as he can, and the boundaries are measured anew. The difference between the indices of the most com-

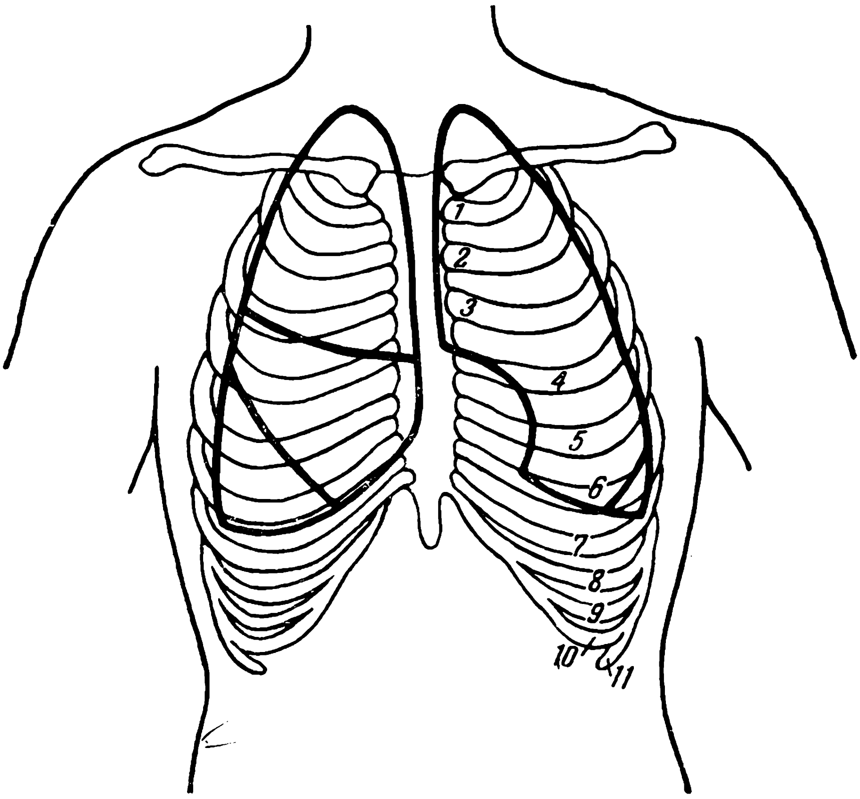


Fig. 22. Lung boundaries (front view)

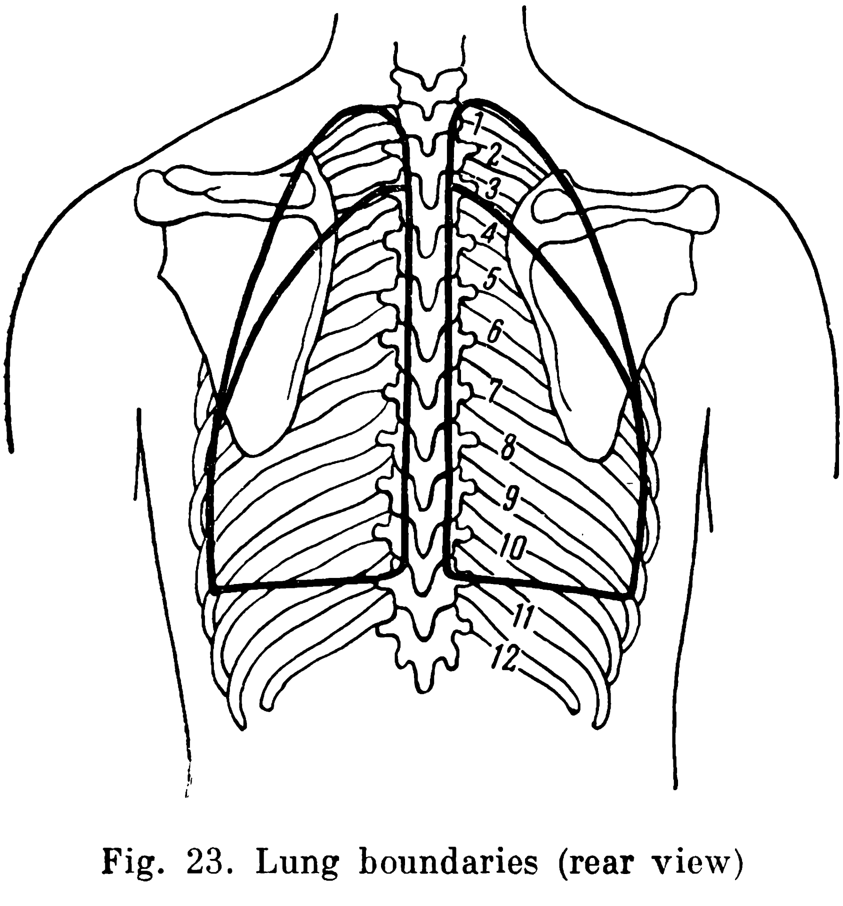


Fig. 23. Lung boundaries (rear view)

plete inspiration and expiration expressed in centimetres indicates lung mobility. Examination of the active mobility of the lungs is undertaken for detecting pathologic changes responsible for a limitation of this mobility. Normally, the active mobility of a lung is 3-4 cm along any line, except the midaxillary, where it is much larger due to the presence of considerable additional spaces in the pleura. Limitation of the active mobility of the lungs is associated with a reduced elasticity of the pulmonary tissue, cicatricial and inflammatory changes in the lungs, and adhesions of the pleurae. The lung margins lose mobility upon a complete adhesion of pleurae, when the pleural cavity is filled with fluid or air, as well as in immobility of the diaphragm caused by various pathologic processes. The respiratory mobility of the lung margins at the apices does not exceed 1 cm.

Comparative percussion. In this type of examination a comparison is drawn between the percussion notes elicited from parts of the thorax lying symmetrically to one another. The patient standing erect, strokes of a uniform force are applied according to a definite scheme: at first, the percussion notes are compared at the lung apices, then in the interscapular space, subscapular region, and on both sides of the anterior surface of the chest.

Normally, percussion notes are not identical in different parts of the thorax. For example, percussion of the lung apices produces a less loud note than that of the lower lobes because of the presence of a thick muscular girdle and a small content of air in the apices. In the second and third intercostal spaces at left the percussion note is somewhat shorter compared with that in the analogous part of the right lung because of the nearness of the heart. Comparative percussion along the midaxillary line, particularly in its lower portions, shows a considerable difference of sounds at right and left owing to the proximity of the liver, which gives out a dull sound upon percussion. Also at left one finds the stomach which gives out a tympanitic percussion note. As a result of this the percussion note at right will be duller than at left. Normally, the tympanitic resonance is to be found only at left along the anterior axillary line downwards, in the so-called Traube's space limited above by the lower lung margin, at right by the left lobe of the liver, at left by the spleen, and below by the rib margin. This space corresponds to the air-containing portion of the stomach, which lends a tympanitic tone to the percussion note. Upon accumulation of fluid in the left pleural cavity tympanitic sounds in Traube's space cease.

Auscultation of the Lungs

Auscultation as a method for examination of patients has been widely used in medical practice since the time of Laennec (1781-1826) who worked out its technique. He also proposed that a hollow wooden tube, now called the stethoscope (from Greek *stethos*—chest and *scopein*—to view), be used for this purpose. Later, stethoscopes began to be made of metal, glass, and ivory. In addition to the conventional stethoscope (Fig. 24), medical practitioners also use Filatov's flexible stethoscope consisting of a hard tube with a flared mouth to be placed over the locality examined and of flexible branches with olives to be inserted into the ears of the listener.

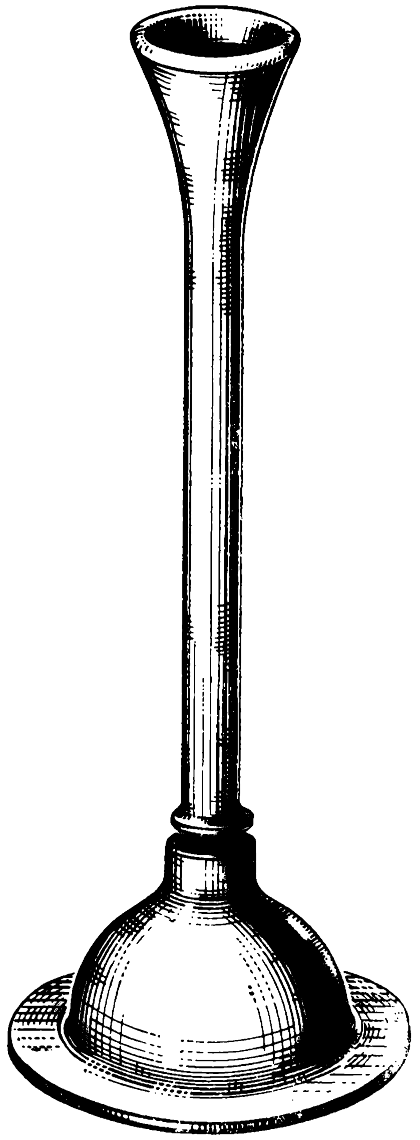


Fig. 24. Stethoscope

The sounds to be listened to are conducted both through the stethoscope walls and through the air inside the tube. Apparently, the stethoscope, while conducting sounds, fails to increase their sonority considerably. The sounds are intensified by the aid of a phonendoscope, i.e., a stethoscope (Figs. 25 and 26) provided with a resonance chamber. Sometimes, a sound-filtering celluloid or ebonite membrane is inserted into the bell-shaped mouth and covered with an animal membrane. This instrument is indispensable in examining severely ill and slovenly patients.

The rules of auscultation. The auscultatory procedure must be carried out in a quiet room. The patient's body should be naked so as to avoid sounds from friction of the clothes. If the body is hairy, the hairs may simulate crepitation; therefore, in such cases they should be moistened or even shaved off. Auscultation may be immediate and mediate. The former is performed by applying the ear directly to the part under examination, the latter by the aid of a stethoscope or phonendoscope. Immediate auscultation is unhygienic. Besides that, in this procedure the examiner hears many sounds coming from an extensive area simultaneously, which makes it difficult to locate a particular sound. It should be also borne in mind that not all localities are feasible for immediate auscultation. In mediate auscultation the stethoscope is applied firmly and perpendicularly to the body surface. To avoid pain being caused to the patient and a decrease in sound vibrations in the locality examined the stethoscope should not be pressed to the latter.

Auscultation is used in examination of the lungs, the cardiovascular system, and sometimes of the organs of the abdominal cavity.

Respiratory murmurs. In auscultation of the lungs in a healthy person one hears a soft respiratory murmur resembling the sound "f" uttered when drawing a breath. This murmur is heard during inspi-

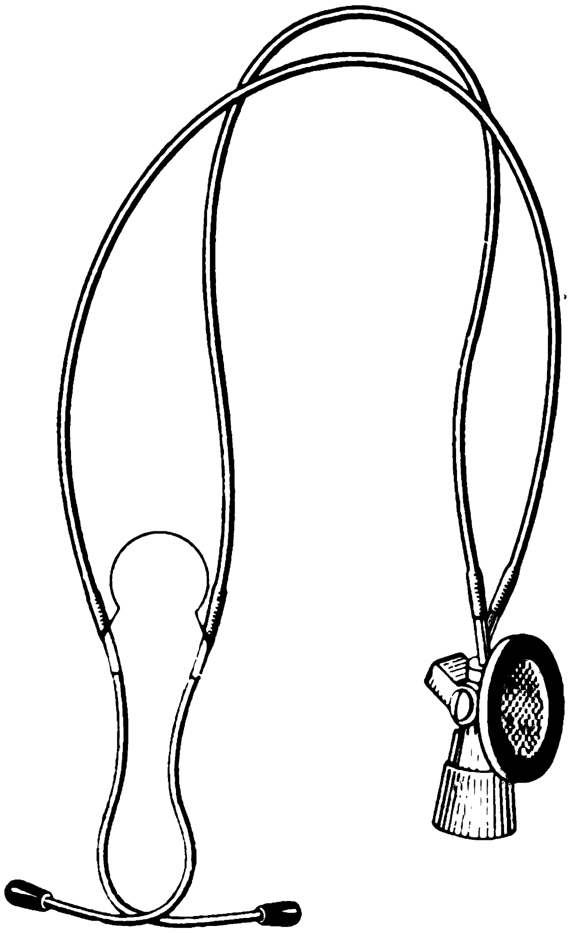


Fig. 25. Phonendoscope

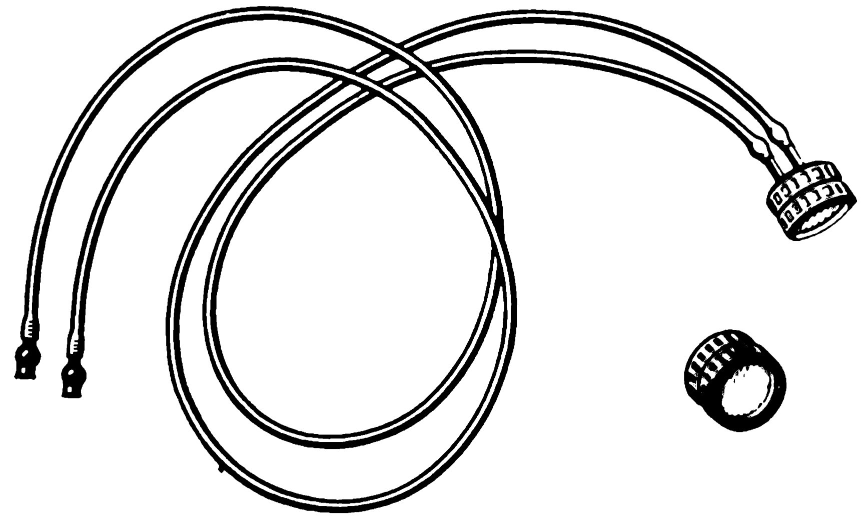


Fig. 26. Phonendoscope

ration. During expiration, no murmur is heard or only a brief sound at the beginning of expiration. It is believed that a normal respiratory murmur is produced in the alveoli by vibrations of their elastic fibres under the action of air entering and distending the collapsed alveoli. That is why this murmur is called vesicular (from Latin *vesicula*, a little bladder).

Vesicular respiration. This respiration is well audible over the front of the chest and in the subscapular regions, particularly in lean persons, and is less distinct at the lung apices. In children under 12-14 years of age, when the thoracic walls are thin and the elastic tension of the lungs is more pronounced, the vesicular respiration is markedly intensified and is called exaggerated or puerile (Latin *puer*—child). Attenuation or cessation of vesicular respiration is seen in persons with shallow breathing, constriction of the larger bronchi, the larynx, and the trachea, in pneumothorax, atelectasis, severe general debility, etc. It is due to a reduction or cessation of air supply to the alveoli. A weakening of vesicular respiration also occurs in conditions leading to a decrease in the sound conduction of tissues, namely, in case of fluid accumulating in the pleural cavity and a collapse or displacement of the lungs from the thoracic wall (in exudative pleurisy), a reduction in the elasticity of pulmonary tissue (in emphysema), in the early period of an infiltrative process, in persons with thick thoracic walls, obesity and a thickened pleura.

The vesicular respiration is intensified in febrile conditions as a result of accelerated respiratory movements.

Prolonged expiration. Prolonged expiration is due to a hindrance to air expulsion during a collapse of the alveoli, constrictions of the bronchioli or bronchi, inflammations or spastic contractions of the latter, as well as weakening of their elastic tension (emphysema). If prolonged expiration is confined to a circumscribed area of the respiratory tract one should above all consider an inflammatory process.

Respiration is called *harsh* when it is coarse in both phases with a prolonged expiration; it is encountered in bronchitis, swelling of the mucous membrane of the bronchi and roughness of their walls.

Saccadic or *interrupted vesicular respiration* is heard over an obstacle in the way of air flow to the alveoli during inspiration, so that the air is supplied in a jerking, irregular manner. It is important diagnostically only when heard over a limited area by reason of some hindrances of an inflammatory character, and is encountered in pulmonary tuberculosis.

Bronchial (tubular) respiration. Normally, bronchial respiration is heard over the larynx, the trachea and the upper part of the interscapular space. It can be reproduced by uttering the sound "kh" while expelling the air with a raised tip of the tongue. The appearance of bronchial respiration points to consolidation of the lung tissue or formation of cavities communicating with the bronchi. A distinct sound of bronchial respiration is due to the fact that in such diseases as tuberculosis and pneumonia affecting considerable areas the vesicular respiration ceases, and one can hear the respiratory murmur of the larynx, trachea, and larger bronchi intensified by the increased sound conduction of consolidated pulmonary tissue. Sometimes, a weak bronchial respiration seems to be heard from a distance; it is found with pulmonary consolidation due to compression with a fluid or tumour. In such cases, a consolidated lung is pushed back, and the examiner hears sounds passing through a thick layer of fluid or tumour tissue.

Amphoric (hollow) respiration. This respiration, which takes its name from the Greek word *amphora*—pitcher, is a variety of bronchial respiration. It is so called because it can be reproduced by blowing air into an empty bottle. It is encountered in patients with pneumothorax, tuberculous caverns, abscesses, if they are located not far from the thoracic wall and are connected to a bronchus or surrounded by consolidated lung tissue.

Mixed respiration. It is engendered in the presence of small foci of consolidation or deep-seated foci covered with normal lung tissue. An inspiration resembles vesicular respiration, and expiration is like bronchial respiration or has a shade of the latter.

Rales. Rales are either dry or moist. *Dry rales* may be sibilant and sonorous. The origin of both the sibilant and sonorous rales is connected with inflammatory processes in the lungs and bronchi as well as with an accumulation in the bronchial tubes of mucus, blood, and fluid resulting from inflammatory oedema. During an inflammation the bronchial mucosa swells and narrows the bronchial tubes with formation of viscid mucus ropes which vibrate like strings under pressure from the passing air. This produces high-pitched treble sounds in the smaller bronchi and low-pitched, sonorous or bass sounds in the larger bronchi. Dry rales occur in bronchitis and are heard both in inspiration and expiration. They may be diffuse and be heard over a wide area in extensive catarrh and bronchial spasm, or limited such as those heard in focal processes in the lungs (tuberculosis, tumour). Dry rales are transitory and sometimes are as loud as to be heard at a distance and felt by the palm pressed to the chest.

Moist rales occur as a rule in bronchi of any calibre due to the passage of air through their inflammatory exudate. They are otherwise known as bubbling rales and may be reproduced by blowing air through a glass tube into a glass of water. In these cases, air bubbles burst, producing a characteristic sound. Small moist bubbling rales are heard in the smaller bronchi, medium bubbling rales in the medium-calibre bronchi, and large bubbling rales in the larger bronchi. Moist rales also differ in brightness of tone. If a bronchus in which a moist rale is heard is surrounded with an air cell tissue the rale will be softened and heard as though from afar. If, however, a bronchus in which a rale is heard is surrounded by a consolidated tissue intensifying sound conduction, the rale will be bright, clear or consonating. Large moist bubbling rales also occur in cavities. Their appearance on the lung periphery points to the presence of cavity because the larger bronchi come short of the periphery. Special importance is attached to small moist bubbling rales; they are heard in pulmonary infarction, tuberculosis and pneumonia. Large bubbling rales occur in caverns or in bronchiectasis. Moist rales are heard in both phases of respiration.

Crepitation results from air passing in inspiration and separating of the alveoli walls as though glued together with an inflammatory exudate. That is why it is heard at the end of inspiration and resembles the sound produced by rubbing hair between the fingers held close to the ear. Crepitation accompanies inflammatory changes in the alveoli, pulmonary oedema, congestion (stasis) in the pulmonary circulation, and pulmonary infarction. Crepitation sounds are more sonorous when the air cells are surrounded by a consolidated lung tissue. Usually, crepitation is first heard in the lower parts of the lungs during the first inspirations because of distention of those alveoli which did not take part in respiration. Crepitation also occurs in debilitated patients compelled to lie on their backs for a long time. Often one has to differentiate between a moist rale and crepitation. In such a case, the physician is guided by the following considerations: moist rales are heard both in inspiration and expiration, whereas crepitation is heard at the height of inspiration. In addition, moist rales change in quality after a cough, whereas crepitation is remarkably permanent.

The pleural friction sound. In healthy persons friction of the smooth pleurae in respiration produces no sounds at all. If they become rough due to an inflammatory process, fibrin deposits or formation of cancerous nodes, as well as an excessive desiccation of the pleurae in consequence of a severe dehydration of the body, there is a pleural friction sound, which varies in tone with the degree of roughness. In an intense roughness the friction sound resembles the crunching of snow under a leather boot or the sound produced by rubbing fresh leather between the fingers; in a slight roughness this friction sound resembles that produced by rubbing a silken fabric. The pleural friction sound is distinctly heard at the sites of the widest respiratory excursions of the lungs, i.e., in the axillary fossa and in the inferolateral parts. A markedly audible pleural friction sound may be not only heard but also felt by the hand applied to the chest. Sometimes, pleural friction sound does not persist and disappears early. It is most often heard in dry pleurisy, in the early stage of wet (excudative) pleurisy, and in the period of exudate resorption. The friction sound is heard in the heart region where the pleural folds come into contact with the pericardium. During extension of the disease process from the pleura to the pericardium there is a pleuro-pericardial friction sound. The pleural friction sound must be differentiated from crepitation. The friction sound is heard at a closer range in both phases of respiration, is intensified on pressure with a stethoscope, applied to the chest and is attended with pain aggravating in a deep inspiration, which is not the case in crepitation.

Bronchophony. One applying his ear to the chest of a healthy person speaking will hear only vague murmuring. If, however, the lung tissue is at places consolidated or contains cavities, one performing auscultation with the ear or by the aid of a stethoscope will understand separate words spoken because the consolidated tissue and air-filled cavity are good sound conductors. The increased resonance of the voice within the bronchi as heard on auscultating the chest is called bronchophony. The latter is determined in the same way as vocal fremitus, that is the patient is told to utter words with many consonants in a low deep voice while his chest is auscultated directly with the ear or by means of a stethoscope. Bronchophony intensifies in patients with lobar pneumonia, tuberculosis, infarction, atelectasis, caverns, cavities, etc., and abates in obstruction of the bronchi and in the presence of pleural adhesions as well as fluid in the pleural cavity.

ACUTE BRONCHITIS (BRONCHITIS ACUTA)

Aetiology and pathogenesis. The cause of acute bronchitis may be inhalation of noxious gases, ammonia, chlorine, phosgene vapours and other war gases, and infectious diseases, such as measles, whooping cough, influenza, pulmonary tuberculosis, typhoid fever and typhus. This disease prevails in spring and autumn with their drastic changes of air temperature. It may affect people of any age, but is liable to run a particularly severe course in children, elderly and senile persons, as well as in persons with a deranged pulmonary circulation.

The leading role in the origination of bronchitis is played by neuroreflex factors. Chilling of the whole or part of the body or irritation of the air passages with chemical substances are responsible for a reflex disturbance in the blood circulation as manifested by hyperaemia of the bronchial mucosa as a result of dilation of blood vessels. This provides favourable conditions for the entry of pathogenic microbes, numerous inhabitants of the upper air passages, into the bronchial mucosa.

Pathologic anatomy. In acute bronchitis, the mucous membrane is swollen, hyperaemic, and velvety in appearance. The bronchial tubes, particularly the smaller ones, become narrower due to abnormal secretions and spasms. Exudate, which is at first scanty, later appears in quantity and acquires a serous, mucopurulent or purulent quality, and in influenza becomes blood-stained. In mild cases, the disease afflicts only the mucous membrane, whereas in a severe condition all layers of the bronchi become involved.

Clinical picture. The clinical picture of acute bronchitis is made up of the general and local manifestations. Among the former the most common are general debility, slight chills, fever, muscle pains in the back and limbs, reduced work capacity, and anorexia (loss of appetite). Of the local symptoms, mention should be made of a scratching and smarting sensation in the throat, which is encountered in concurrent tracheitis and laryngitis, coughing with a mucous, seromucous or mucopurulent discharge, and pain in the chest. Acute bronchitis lasts from several days to a fortnight.

Examination of the patient, percussion and palpation of the thorax reveals no significant abnormalities. A somewhat harsh respiration is heard on ascultation as a result of the swelling of the bronchial mucosa, expiration is prolonged as compared with inspiration, respiration is weakened due to obstruction of a bronchus with mucus. Dry rales are heard, which are sonorous in affection of the larger, and sibilant in affection of the smaller, bronchi. In the presence of profuse and liquid exudation from the bronchial mucosa, moist rales may also be heard, both small bubbling and large bubbling ones. In debilitated persons the disease process sometimes extends to the interstitial and alveolar lung tissue and causes bronchopneumonia, which is characterized by a sudden elevation of temperature, rapid breathing, deterioration of the general condition, dull sounds elicited on percussion from the affected area, and a large number of sonorous moist rales heard on auscultation, as well as cyanosis.

Treatment. A patient with fever must be confined to bed and given aspirin or pyramidon per os in doses of 0.5 g three times daily, much lukewarm liquid to drink, and raspberry tea. For chest pains, revulsive remedies, such as cupping glasses, mustard and turpentine plasters applied to the chest, as well as hot baths for the feet are recommended. For a very obnoxious cough the patient is given codeine (formulas Nos. 56, 57), dionine (formulas Nos. 58, 59). In case of a difficulty in expectoration of phlegm, thermopsis (formula No. 60) or ipecacuanha (formula No. 61) are used. In tracheitis and laryngitis attending bronchitis one is recommended to drink mineral water (half-a-glassful) with warm milk or milk with cooking soda (one teaspoonful per glassful of milk). If bronchial spasm is also present, ephedrine or theophedrine are prescribed.

CHRONIC BRONCHITIS (BRONCHITIS CHRONICA)

Aetiology and pathogenesis. This disease occurs in chronic infections, after acute bronchitis, prolonged irritations of the air passages with dust, nicotine, abuse of alcoholic drinks, as well as in chronic diseases of the heart and lungs. Chronic bronchitis also occurs in persons exposed to occupational dust hazards, i.e., workers of the tobacco and porcelain factories, flour mills, colieries, etc.

Pathologic anatomy. The bronchial mucosa is usually swollen and congested, with a copious exudation. The disease process in the bronchi is of a productive character, but later atrophic changes and fibrinous proliferations come on the stage; the latter invade all the layers of the bronchi, leading to a thinning of the bronchial walls and formation of bronchiectasis.

Clinical picture. Patients complain of a dry or moist cough which becomes worse at night and in the morning, as well as in a wet and cold weather. In lesions of the bronchioles the cough is particularly obnoxious. The body temperature is usually normal, but in exacerbations may rise to 37.5-38°C. No objective changes are discovered on percussion. Auscultation, however, reveals a harsh respiration, prolonged expiration, dry rales and a small number of moist rales of a variable pitch.

Several varieties of chronic bronchitis have been described. For example, this disease is sometimes accompanied by a thick sputum discharge with dense adhesive strands and fibrin resembling casts of the bronchial lumen in shape. This condition is known as fibrinous bronchitis. Putrid or foetid bronchitis is a variety characterized by the discharge of an offensive sputum containing Dittrich's plugs with a large number of streptococci and spindle-shaped bacilli.

Chronic bronchitis lasts for years. The bronchial walls, as a result of the growth of connective tissue and loss of elasticity, tend to dilate under the action of coughing and bronchiectasis develops. The onset of bronchiectasis is facilitated by the inflammatory process extending to the tissue surrounding a bronchus, where fibrosis develops also.

Treatment and prophylaxis. Treatment prescribed for an exacerbation of chronic bronchitis is the same as that for acute bronchitis. Of the symptomatic remedies for a difficult expectoration of sputum, *Thermopsis* and ipecacuanha are recommended. For an effective control of chronic bronchitis it is necessary to eliminate harmful environmental factors contributing to the occurrence of acute bronchitis: dust, chilling, chemical irritants, smoking, addiction to alcohol. Measures should be taken against all diseases likely to provoke the onset of chronic bronchitis, asthma in particular, as well as inflammatory lesions in the upper air passages. A good effect is produced by staying outdoors in a dry weather for a long time. Climatic therapy in a dry mountainous locality and measures to improve the general condition of the nasopharynx are advisable.

BRONCHIAL ASTHMA (ASTHMA BRONCHIALE)

Bronchial asthma manifests itself by paroxysmal attacks of dyspnoea due to a disturbance in bronchial patency.

It was described by Hippocrates, G. Sokolsky, and S. Botkin. The latter attributed attacks of asthma to spasms of the smooth muscles of the bronchi. Bronchial asthma is widely prevalent in all countries: in the USA about 3 per cent and in England and Wales 0.9 per cent of the population are affected with bronchial asthma.

Aetiology and pathogenesis. In the mechanism by which an attack of dyspnoea makes its appearance, an increase in the tone and relaxation of the sympathetic nerve both play a role. This results in a spasm of the bronchial smooth muscles and bronchial narrowing with a copious mucous secretion from the bronchial mucosa and bronchial oedema responsible for laboured breathing. Evidence in favour of the above-described mechanism of initiation of bronchial asthma is the fact that an injection of adrenaline stimulating the sympathetic nervous system and of atropine paralyzing the vagus nerve endings aborts an attack.

The cause of the heightened excitability of the parasympathetic (vagus nerve) and relaxation of the sympathetic nervous system is ascribed to different factors by different authors. Some speak out in favour of the allergic nature of bronchial asthma, i.e., hypersensitivity of patients to a number of substances of protein and vegetable nature (allergens), whose entrance into the body, even in a negligible quantity, provokes an allergic reaction. Others adhere to the neurogenous theory. The reasons for the allergic theory of asthma were drawn from clinical observations in which asthma was seen to have occurred after inhaling the odours of perfumes, flowers, ursol (in furriers), ipecacuanha (in druggists), upon contact with newly-mowed hay (hay fever), as well as after eating certain species of fish, crayfish, eggs, strawberries, etc. The allergic nature of bronchial asthma is also proved by its occurrence simultaneously with other allergic diseases: urticaria, eczema, Quinke's oedema, which is evidence in favour of allergens being nonspecific. Therefore, one and the same allergen may give rise to different allergic diseases: in some cases, bronchial asthma, in others, urticaria, or both diseases simultaneously. The allergic nature of bronchial asthma is corroborated by cases of recovery from bronchial asthma after withdrawing allergens responsible for its attack.

For an allergic reaction to become manifest, a preliminary sensitization of the body is necessary, either congenital or acquired, that may provoke attacks of asthma only after a prolonged exposure to allergens to which a patient is hypersensitive.

There are also quite a few followers of the neurogenous theory of bronchial asthma. Attacks of asthma due to emotional strain—a disorder in the vegetative nervous system, or in persons with craniocerebral injuries as well as attacks initiated by reflex stimuli coming from a deviated nasal septum, an inflamed gallbladder, nasal polyps, etc., give one reason to regard bronchial asthma as psychoneurosis.

The course of bronchial asthma is greatly influenced by infections. Pneumonia, influenza, tonsillitis, maxillary sinusitis (highmoritis), and other infective foci in the body provide favourable conditions for progression of allergies, sensitization of the body with metabolic products of the pathogenic flora and protein decomposition in the foci of infection. The latter in their turn become a source of pathologic reflexes.

A natural association has been noted between the state of the endocrine system and the course of bronchial asthma (pubescence, menstruation, pregnancy, lactation, etc.). Among the endocrine glands emphasis should be laid on the adrenals, which play an important role in sensitization of the body. This is proved by the favourable influence of cortisone, prednisolone and ACTH (adrenocorticotropic hormone of the anterior lobe of the pituitary body) on the course of bronchial asthma.

Pathologic anatomy. Patients with bronchial asthma suffer from pulmonary emphysema. There is mucus in the smaller and medium-sized bronchi, the bronchial walls are thickened and the bronchial tube contains eosinophils and Charcot-Leyden crystals: the alveoli are irregular, distended, their walls are thinned, and the division-walls between them are at places ruptured. There is hypertrophy and dilatation of the right cardiac ventricle.

Clinical picture. Attacks of asthma occur of a sudden, usually at night. Sometimes, they are preceded by a sense of constriction in the chest, general weakness, drowsiness, yawning, a tickling sensation in and obstruction of the nose. In patients with food allergy nausea, heartburn, etc., are noted among the prodromal symptoms.

During an attack the patient usually takes a sitting posture, leaning on some hard object with his hands to ease respiration. He feels a great strain in the auxiliary muscles: the scalenus, the sternocleidomastoideus, and the muscles of the abdominal press (prelum abdominale). Not infrequently, during an attack the patient jumps out of bed and opens a window so as to draw a breath of fresh air. He looks frightened, with a pale face and profuse perspiration. His breathing is slow and difficult, mainly because of wheezing expiration which is heard even from a distance. The body temperature is normal or slightly elevated. The veins on the neck are swollen, the pulse rate is slowed down, and the arterial pressure is lowered. The thoracic cage becomes inflated, as though fixed at inspiration. The percussion note has a tympanitic tone; the lower boundaries of the lungs are depressed, and their excursion is limited. There is acute emphysema. The absolute cardiac dullness is undetectable because of pulmonary inflation. A large number of sibilant and crepitant rales are heard on auscultation over the lungs.

The duration and gravity of an attack vary in different persons and even in one and the same person in different periods of disease. An attack lasts from several minutes to a few days. It begins to abate with a cough, at first accompanied by a scanty discharge of viscid, vitreous sputum, which later becomes mucopurulent. After an attack the patient falls asleep and his general condition rapidly improves in cases of fresh cases of bronchial asthma not complicated by diffuse bronchitis, emphysema, or pneumosclerosis.

In certain cases, an attack fails to terminate within the first few hours and continues for days and weeks, while the sputum discharge gives no relief. Such attacks are known as status asthmaticus. Attacks of bronchial asthma may recur in a definite sequence. At first they are infrequent. Sometimes they recur every month, or week or become continuous, and in time cease to run their characteristic course, being manifested only by chronic bronchitis which becomes worse in spring and autumn. Attacks of bronchial asthma due to allergens may later be provoked by physical or emotional strain, a cold wind, and various odoriferous substances. Climate and meteorologic conditions are also factors important for the origination and course of bronchial asthma. Cyclones contribute to a greater frequency of attacks, whereas at an altitude of 1,000-1,200 metres above sea level attacks are rare or absent altogether.

In the typical patient, bronchial asthma is identified easily. Breathlessness with laboured expiration, acute pulmonary emphysema, the presence of Curschmann's spirals and Charcot-Leyden's crystals in sputum, and eosinophilia make the diagnosis certain (Fig. 27).

The differential diagnosis should be established between cardiac, symptomatic, and hysterical asthma. A cardiovascular disorder (hypertensive vascular disease, myocardial infarction, cardiosclerosis, etc.), enlargement of the left cardiac ventricle, stasis in the pulmonary circulation and in the liver, the absence of an effect from the administration of adrenalin, as well as pulmonary oedema with a considerable number of moist rales and sometimes with a pink, frothy sputum discharge are all symptoms ruling out bronchial asthma. Symptomatic asthma is seen with pneumosclerosis and pulmonary emphysema. In hysterical asthma, respiration is shallow and quick, with no objective signs of changes in the lungs, sputum, and blood, i.e., with the absence of emphysema and sibilant pulmonary rales of variable calibre during an attack, laboured expiration, vitreous sputum, and eosinophilia.

The *prognosis* depends on the frequency of attacks and the degree of changes in the lungs and in the nervous system. Bronchial asthma persisting for many years may result in pulmonary emphysema, chronic bronchitis, pneumosclerosis and cardiopulmonary insuffi-

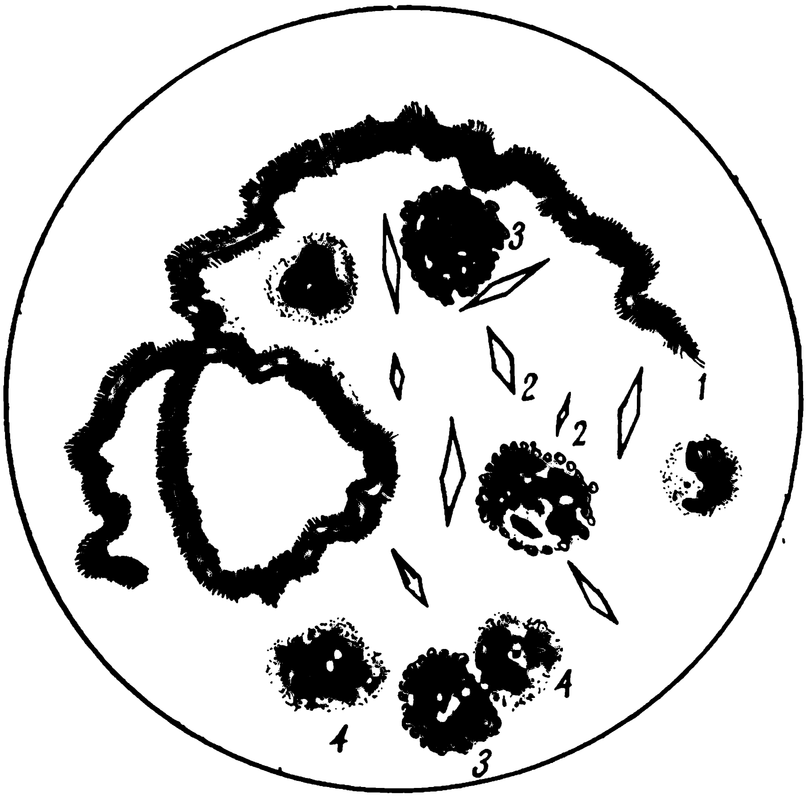


Fig. 27. Sputum in bronchial asthma
1—Curschmann's spiral; 2—Charcot-Leyden's crystals; 3—eosinophils; 4—neutrophils

ciency, and may end in death from complications it has produced. Death during an attack occurs very seldom, mainly in elderly persons. Patients liable to rare attacks and without concurrent complications are perfectly fit for working. Those suffering from frequent attacks with extensive changes in the respiratory and circulatory organs have a limited capacity for work or are totally disabled.

Treatment and prophylaxis. An attack of bronchial asthma is best aborted by a subcutaneous injection of a 0.1 per cent adrenalin hydrochlorate solution (formula No. 36) in a dose of 0.5-1.0 ml, which stimulates the sympathetic nervous system and relieves the spasm of the bronchial smooth muscles in a matter of 2-3 minutes, simultaneously paralyzing the vagus nerve endings. The action of adrenalin lasts not longer than 1-2 hours, therefore, in a prolonged attack, repeated injections (up to 8-10 daily) may be required. Adrenalin is contraindicated in the following cases: (a) hypersensitivity to adrenalin manifested by headaches, trembling (tremor), excessive palpitation (tachycardia); (b) hypertensive vascular disease, stenocardia, cardiovascular insufficiency.

Some of the recent remedies used in clinical practice are noradrenaline, isoprenaline, and euspiran.

A weaker, yet more lasting effect is produced by ephedrine, an adrenalin preparation (formula No. 30). When administered per os (0.025 g doses in tablet form) it is active during 4-6 hours; a subcutaneous injection gives an effect not earlier than in half-an-hour. Ephedrine sometimes causes untoward effects (insomnia, dysuria), which can be eliminated by a simultaneous administration of dimedrol (formula No. 173), luminal (formula No. 110), etc. Ephedrine is commonly prescribed for moderately severe bronchial asthma as well as to prevent the onset of an attack. An attack of bronchial asthma can also be aborted by administration of theophedrine and the Czechoslovak preparation antasthman, which consists of thornapple, henbane, and belladonna containing atropine as well as alkaloids related to them moistened with a 10 per cent potassium nitrate solution. Half-a-teaspoonful of this preparation is burnt and the smoke inhaled.

Another remedy against attacks of bronchial asthma is a 0.1 per cent atropine sulfate solution (formula No. 35) administered in a dose of 1-2 ml. Its application is indispensable when asthma is concurrent with stenocardia, coronary sclerosis, circulatory disturbance, as well as cardiopulmonary insufficiency. Atropine produces a paralyzing effect on the vagus nerve endings and relaxes the bronchial smooth muscles, causes a dry sensation in the mouth, and a visual disorder. Platyphyllin (0.002-0.006 g) which is prescribed for the same purpose instead of atropine gives no side-effects.

During the past few years, bronchial asthma has been extensively treated with hormone drugs—cortisone, prednisolone, prednisone, and ACTH—which often give an effective cure in a severe asthmatic condition. ACTH is injected intramuscularly in doses of 10-20 units 2-3 times daily, cortisone in doses of 100 mg daily, and prednisone and prednisolone in 25 mg doses. Hormone therapy is contraindicated in the presence of concurrent atherosclerosis, hypertensive vascular disease, and peptic ulcer.

For mild forms of bronchial asthma, khellin may be prescribed in doses of 0.02 g to be taken twice daily, paraverine in doses of 0.03 g, and tiphen in doses of 0.015 g to be taken 2-3 times daily. Subcutaneous injections of I. Traskov's and M. Skrypnik's fluid are used as a spasmolytic as well as antiasthmatic measure.

Euphyllin (formulas Nos. 26, 26*a*, and 27) is also used for bronchial asthma. It produces a strong dilative effect on the bronchi and the renal and coronary vessels; therefore, it is indispensable where asthma combines with stenocardia or coronary sclerosis. Euphyllin should be injected intravenously in 20 ml of a 40 per cent glucose solution at a slow rate. It is sometimes administered in suppository form. Euphyllin substitutes may also be used, for example, diaphyllin administered intramuscularly (1 ml of 24 per cent diaphyllini gluteosi) or intravenously (5 ml of 48 per cent diaphyllini venos). In cases where attacks of bronchial asthma are coupled with sinusitis, tonsillitis, maxillary sinusitis (highmoritis), pneumonia or frequent exacerbations of chronic bronchitis, antibiotic therapy mitigates its course. Effective in such cases is penicillin administered in aerosol form in a dose of 100,000-200,000 units in 2 ml of physiologic salt solution once or twice daily. A vaccine prepared from the patient's sputum is also recommended. During an attack of bronchial asthma, injections of narcotic drugs, such as morphine, pantopon, etc., are contraindicated since the latter are liable to suppress the respiratory centre and may cause asphyxia and death. An enema of 2 g of chloral hydrate in 20 ml of physiologic salt solution may also abort an attack sometimes. Sleep therapy is contraindicated because in a sleeping person the vagus nerve tone is increased, and an attack of bronchial asthma may be provoked. During an attack the patient's condition is relieved with mustard plasters and cupping glasses applied to the chest, and in the off period with expectorants—thermopsis (formula No. 60), ammonium chloride (formula No. 44), alkalis, as well as potassium iodide (formula No. 190) which serves to liquefy sputum.

A radical therapeutic measure against bronchial asthma is reduction of the body reactivity. For this purpose, the patient's mode of life is normalized, his nervous system fortified by means of sedatives and roborants, bromides, luminal, hydrotherapy (water cure), psychotherapy, respiratory exercises, sports and physical culture. At the same time, it is necessary to eliminate those allergens to which the patient is known to be hypersensitive, for which purpose his occupation and job may be changed. Where no allergens have been revealed, it is wise to undertake protein therapy, tissue and shock therapy, hyperthermia, treatment with honey bee venom, and acupuncture. Sometimes, attacks of bronchial asthma cease after the sufferer has moved to live in another climate. It should be borne in mind at the same time that some patients feel better in mountainous localities, others on a sea coast. Treatment at a health resort is also helpful.

BRONCHIECTASIS (BRONCHIECTASIA)

Bronchiectasis (Latin *bronchus* and *ectasia*—distention) is a chronic condition with a deep involvement of the bronchopulmonary apparatus and various complications.

The disease affects persons of any, mostly advanced, age.

Aetiology and pathogenesis. Bronchiectasis may be congenital and acquired. The former develops during foetal life as a result of malformations in the bronchi and atelectasis of the lungs. Acquired bronchiectasis is encountered in persons of any age, but most commonly is consequent upon measles, whooping cough, bronchitis, pneumonia, pleurisy, tuberculosis, syphilis, pneumoconiosis, war gas poisoning, bullet wounds of the chest, and inflammatory processes in the peribronchial tissue.

In the majority of cases, bronchiectasis follows pneumonia. In addition to the alveoli, the surrounding tissue also become involved in the disease process. When its outcome is unfavourable, the inflammatory process in the interstitial tissue leads to the formation of scar tissue surrounding the alveoli and bronchi. In such cases, the blood and lymph circulation in the bronchi are disturbed and the latter show symptoms of congestion with swelling of the mucous membrane. A marked impairment in the trophic condition of the walls entails degeneration of the elastic muscle tissue of the bronchial wall into connective tissue and the development of secondary bronchitis.

Various processes of an inflammatory, mechanical and toxic character occurring in the lungs and pleura give rise to pneumosclerosis, which may be circumscribed or diffuse, with symptoms of bronchitis and peribronchitis, as a result of which the bronchi lose their tone, become pliant and gradually dilate. Distention of the bronchi may also be accounted for by a loss of elasticity of the pulmonary tissue in the affected region. The elastic tissue of the intact regions of the lung distends the altered tissue and the bronchi contained therein.

Bronchiectasis most commonly develops in the left lung, primarily in the lower lobes and very rarely in the upper lobes. This is due to the left bronchus being narrower than the right, as well as to its branching off at an acute angle and forming a physiologic narrowing where the left branch of the pulmonary artery bends over it. The localization of bronchiectasis in the lower lobes is explained by poor drainage conditions in the latter.

Pathologic anatomy. Bronchiectasis may be cylindrical with a uniform dilatation of the bronchial tube, sacculated, i.e., with a limited prominence of certain parts, fusiform (spindle-shaped), which is a combination of the above two forms, and varicose, shaped as rosaries. The bronchial mucosa is inflamed and congested, and sometimes ulcerated. In the majority of cases, a dilated bronchus is not larger than 1-2 cm in diameter, yet one is likely to meet with smaller (the size of a pea) and larger (the size of an apple) dilatations.

Clinical picture. In the early period of the disease, the common complaint is one of coughing with sputum discharge, particularly in a wet weather. Many sibilant (whistling, hissing) rales are heard on ausculatation. This period may last for years. The patient is very liable to develop pneumonia, exacerbations of chronic bronchitis, and pleurisy. The sputum discharge is mucopurulent and is usually not profuse.

In the second period, the disease manifests itself with a paroxysm of coughing, primarily in the morning, with as much discharge as to fill the mouth, the copious and foetid mucopurulent sputum sometimes streaked with blood. Sputum secretion is easily provoked by a change in posture, especially so when the body is tilted forward. The amount of sputum discharged in 24 hours ranges from 0.5 to 1 litre. The sputum has three layers and no elastic fibres.

In this period, bronchiectasis is characterized by frequent haemoptysis (blood-spitting), the sputum ranging in quality from blood-streaked to profusely blood-stained. There are, however, special forms of this disease when no cough with sputum discharge occurs for a long time, and the cardinal symptom is periodically recurring pulmonary haemorrhage and haemoptysis. These are the so-called dry forms of bronchiectasis.

The sputum on bacteriologic examination shows the presence of pneumococci, various types of streptococci, staphyllococci, Pfeiffer's and Friedlaender's bacilli, and other organisms.

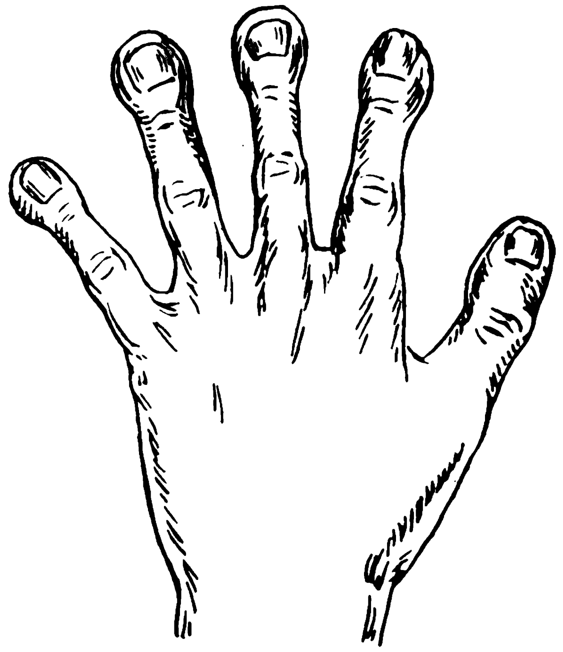


Fig. 28. Clubbed fingers

The body temperature rises considerably in retention of sputum as well as in exacerbations of catarrhal symptoms in the lungs, and persists at a high level until the process has abated. An elevation of temperature is accompanied by dyspnoea and cyanosis. In far advanced cases, there is emaciation, while the face is pale, puffy, and cyanotic. The fingers and sometimes toes are clubbed (Fig. 28), while the nails acquire the form of watchglasses.

The findings of percussion and auscultation of the chest depend on the degree and depth of localization of changes in the bronchi and lungs. The chest becomes barrel-shaped. On percussion of the affected side, a dullness of the percussion note is determinable against the general background of bandbox sounds, and becomes especially audible over massive superimposed fibrosopleural lesions and perifocal inflammatory infiltrations. Following sputum expectoration, tympanitis may be discovered in extensive bronchiectasis. The findings obtained on auscultation of the chest depend on the coexistent pulmonary and pleural affections, their extension, localization, and phase of development. A weakened, harsh, less often bronchial, respiratory sound is usually heard. Especially characteristic are moist rales of a variable calibre, which are heard for years over one and the same region, most commonly in the lower parts of the left lung. The number and character of rales change in proportion to the degree of bronchial obstruction. In the morning rales are usually greater in number than later in the day.

A moderate neutrophilic leucocytosis with a leftward shift in the differential cell count, and elevation of the ESR are observed in the blood in exacerbations and complications of the disease process. Upon abatement and termination of an exacerbation, the blood condition returns to normal.

The bronchi filled with a contrast substance (iodolipol) give the characteristic x-ray picture of a "tree with leaves" (Fig. 29) as distinct from healthy bronchi which look like a "tree without leaves".

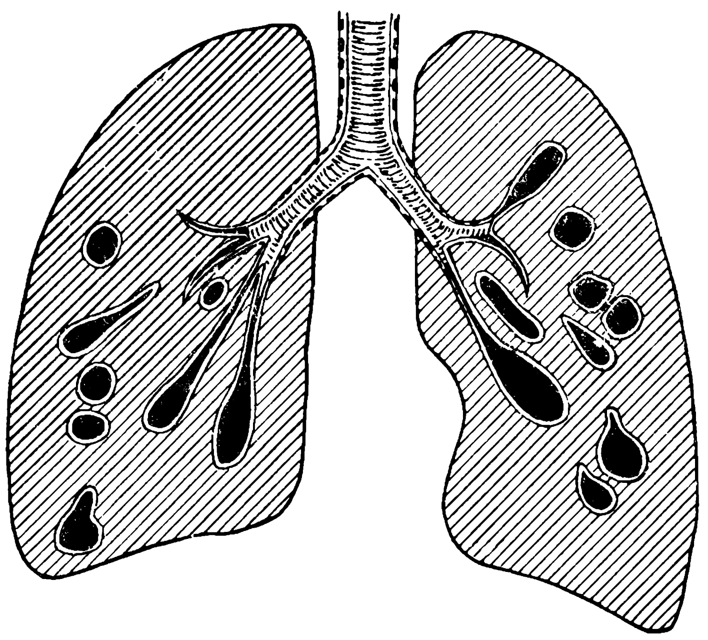


Fig. 29. Multiple bronchiectases

The condition of the cardiovascular system in the early period of the disease is quite satisfactory. Later, the patient complains of palpitation of the heart and breathlessness. He develops oedema of the lower extremities, enlargement of the liver, swelling of the veins on the neck, and ascitis. All these abnormalities are associated with cardiopulmonary insufficiency which comes on gradually. The latter also results from changes occurring in the alveoli and capillaries of the pulmonary circulation due to pneumosclerosis and pulmonary emphysema, these inevitable attendants on bronchiectasis. In pneumosclerosis, part of the alveoli with capillaries lodged within them undergo destruction, while another part of the alveoli become distended and cause a marked narrowing of the capillaries which traverse the walls of the alveoli.

The first symptom is respiratory insufficiency as manifested by breathlessness and general cyanosis. At the same time, collateral circulation comes into being through the arteriovenous communications, and part of the blood from the pulmonary artery, through anastomoses and viens of the bronchi, is supplied to the pulmonary veins, bypassing the pulmonary circulation and remaining inadequately oxygenated. Impairment of lung respiration in this period may be ascertained by respiratory function tests. A test with restrained breath shows a reduction from 30-40 seconds to 20 seconds and less; the vital capacity of the lungs is reduced from 3.5-4 litres to 2-2.5 litres, and oxygen saturation is reduced from 97 to 91 per cent.

Later, symptoms of cardiac insufficiency appear, which is at first confined to the right ventricle and then becomes total.

The increased work of the heart because of hypertension in the pulmonary circulation gives rise to hypertrophy and distention of the right ventricle.

Functional changes in the lungs and heart may be conventionally subdivided into the following three stages: (a) latent; (b) pulmonary insufficiency; (c) cardiopulmonary insufficiency. In the first stage, the patient complains of a cough with sputum discharge and breathlessness on exertion due to oxygen deficiency; in pulmonary insufficiency, the existing symptoms are joined by frequent perifocal pneumonia. In this period, a great number of rales are heard over the lungs, and cyanosis is present as a result of oxygen starvation, which, as distinct from cardiac cyanosis, extends throughout the body, while the extremities remain warm. In the final stage, cardiopulmonary insufficiency is manifested by swollen veins on the neck, enlargement of the liver, oedema, ascitis, etc.

The third stage is characterized by a further advancement of the disease process with a considerable general intoxication, emaciation, and severe complications, such as amyloidosis, amyloid nephrosis, septicopyaemia, a metastatic brain abscess, etc., which sometimes end fatally even before cor pulmonale comes on the stage.

It is necessary to differentiate bronchiectasis from a lung abscess and tuberculosis. In bronchiectasis, the sputum discharged contains no tuberculous mycobacteria, the disease process is confined primarily to the lower parts of the lungs, while the x-ray picture, as distinct from that of tuberculosis, is quite scant although considerable changes are revealed on auscultation. There are symptoms of clubbed fingers and curvature of the nails over the finger ends, the tuberculin test is negative, and no foetid sputum discharge is present to indicate a purulent dissolution of the pulmonary tissue. The differential diagnosis between bronchiectasis and lung abscess involves a number of difficulties, particularly when bronchiectasis is complicated by an abscess. A certain clue may be given by a considerable increase in the number of leucocytes with neutrophilia and a leftward shift in the differential blood count, the presence of a two-layered sputum with elastic fibers, and an x-ray examination: a cavity displaying a level of liquid is characteristic mainly of a lung abscess.

Prognosis is ominous, and chances of recovery are slight. The degree of work capacity varies with the stage of the disease. In the early period it may be retained, in the later stages markedly reduced. Death may be caused by cardiovascular insufficiency, amyloid degeneration of the kidney attended with uraemia, metastases into the brain, or development of carcinoma from a dilated bronchus.

Treatment. The patient should above all be provided with hygienic conditions, i.e., be lodged in a well-ventilated and dry room, and put on a diet of high caloricity rich in animal proteins and vitamins, particularly vitamins A and C. In the period of exacerbation, hospitalization or confinement to bed at home is advisable. The bronchi are cleansed of pus by means of putting the patient in Quincke's posture (drainage by posture), aspiration through a bronchoscope, in combination with bronchial lavage (irrigation) followed by an intrabronchial administration of antibiotics (streptomycin, penicillin) in a dose of 100,000-200,000 units. Antibiotics may be administered intratracheally, through a catheter. Simultaneously, antibiotic aerosol inhalations may be employed. In some cases, the application of sulpha drugs in combination with antibiotics is highly efficacious.

The previously used methods of therapy with thirst, novarsenol, alcohol, sodium benzoate, and x-rays have been abandoned in view of their inadequate efficacy. Treatment at a health resort is given only in the initial stage of bronchiectasis. It is advisable to carry out treatment in steppe areas with a dry and warm climate. In the presence of segmental and unilateral lesions of the lungs surgery may be helpful.

Prophylaxis consists in measures of control of pneumonia and diseases of the upper air passages as well as pulmonary atelectasis as a sourse of bronchiectasis. Radical surgery is recommended in sinusitis and tonsillitis, as well as respiratory exercises, general invigorating therapy, and hardening of the body.

PULMONARY EMPHYSEMA (EMPHYSEMA PULMONUM)

Pulmonary emphysema (Gr. *emphysema*—inflation) develops in the following cases: (a) in acute expansion of the lungs due to prolonged respiratory exercises; (b) respiratory failure in the whole or part of a lung attended with a compensatory expansion of the other lung; (c) breathing with laboured expiration (in bronchial asthma, pneumonia, prolonged cough, etc). In such cases, the increase in the pulmonary volume and the air content of the lungs are not associated with anatomic structural changes in the respiratory apparatus, and are merely functional in character. The action of factors reducing the elasticity of the lung tissue, however, may be responsible for a persistent organic lesion and chronic alveolar emphysema of the lungs. The latter is fairly frequent, particularly in men above 50 years of age, in whom its rate is two or three times that in women.

Aetiology and pathogenesis. Pulmonary emphysema is due to the following causes: (a) bronchitis, tracheitis, especially recurrent ones, pneumonia, bronchiectasis, pulmonary tuberculosis, persistent stasis in the pulmonary circulation, diffuse pneumosclerosis, pneumoconiosis, extension of an inflammation to the interstitial tissue, lung lesions produced by asphyxiating and intoxicating agents, etc.; (b) various diseases liable to throw a strain on the respiratory function which is the case in bronchial asthma. In the early stages, bronchial asthma causes nothing but an acute distention of the lungs which passes after the termination of an attack. Later, the distention becomes stubborn. An aetiologic role in chronic emphysema is played by coughing, particularly in chronic bronchitis and pneumosclerosis, which tend to heighten the intrabronchial and intra-alveolar pressure. Among the factors contributing to the onset of emphysema are all occupations involving physical exertion and intensified respiration (glass-blowers, singers, players on brass-wind instruments, loaders, etc.). Emphysema arises in acute or chronic inflammatory processes or congenital instability of the elastic lung tissue associated with ageing. An important role in the development of chronic emphysema is also played by neurogenous factors causing disorders in the muscular tone of the bronchi, bronchioles, changes in the bony and cartilaginous structures of the thorax (ossification of costal cartilages, kyphosis of the vertebral column), as well as congenital weakness of the elastic elements of the lungs.

One must discriminate between the two varieties of pulmonary emphysema: senile and obstructive. In senile and elderly persons pulmonary emphysema results from impairment of the elastic qualities of the lung tissue irrespective of the patient's past history of respiratory diseases. In such cases, there is no lung expansion, no hypertrophy of the right cardiac ventricle and no disorder in gas exchange.

The most important is obstructive pulmonary emphysema, which results from bronchial asthma, pneumonia, bronchitis, bronchiolitis, and pneumosclerosis. It is accompanied by destructive and atrophic changes in the lung tissue, massive destruction of capillaries, alveoli and interalveolar walls, with a persistent inflation of the lungs which interferes with expiration, lung ventilation and normal gas exchange. The residual air volume is considerably increased, and is attended with hypertrophy of the right cardiac ventricle because of an increased pressure in the pulmonary circulation.

The most formidable complication of pulmonary emphysema is respiratory insufficiency, developing as a result of arterial oxygen unsaturation (hypoxaemia) and retention of carbon dioxide in the blood (hypercapnia). Gas exchange, however, is not disturbed immediately. Early in the disease process, gas exchange is supported at the normal level owing to the compensatory mechanisms (erythrocytosis, haemoglobin increase, intensified lung ventilation, and more vigorous contraction of the right heart). Subsequently, with the progressive aggravation of the functional and organic changes in the lungs and a diminution of the respiratory surface, oxygen deficiency—anoxoemia and hypercapnia—come on the stage.

Pulmonary emphysema is known to occur in three stages: (a) initial; (b) active, and (c) severe.

In the initial stage of pulmonary emphysema there is good compensation owing to the moderately intensified respiration and pulmonary ventilation. The oxygenation of the arterial blood reaches 95 per cent. The right cardiac ventricle is moderately enlarged, and the amount of residual air in the lung is increased insignificantly.

In the active stage of pulmonary emphysema oxygenation does not exceed 90 per cent, the cardiac output is increased, the vital capacity of the lungs is reduced to 70 per cent, while the amount of residual air in them is increased considerably.

In the severe stage there is respiratory difficulty, the oxygenation of the blood does not exceed 70 per cent, the bloodflow rate is decelerated, the venous pressure is heightened, and there are signs of congestion both in the pulmonary circulation and in the veins of the systemic circulation, and ascitis. Cardiovascular insufficiency known as pulmonary heart disease (cor pulmonale) sets in.

Pathologic anatomy. At autopsy incision of the pleura is not followed by a collapse of the lungs because their elasticity is lost and the bronchi are occluded with secretions. Under the pleura, air blebs caused by coalescence of alveoli are determinable sometimes, a condition known as *bullous emphysema.* On section, the lungs are anaemic and a mucopurulent mass is pressed out of the bronchi. Microscopy reveals atrophy of the interalveolar division walls (septa), coalescence of alveoli, growth of fibrous tissue along the course of the bronchi and blood vessels (pneumosclerosis), areas of bronchiectasis, inflammatory symptoms in the bronchi, and hypertrophy of the right cardiac ventricle.

Clinical picture. Patients complain of breathlessness with laboured expiration and coughing. The respiratory movements are aided by the auxiliary respiratory muscles. The force of expiration is reduced to such an extent that the sufferer is unable to blow out a candle flame. The face is puffy and cyanotic, the neck is short and with swollen veins. The thoracic cage is emphysematous; it is short, barrel-shaped and enlarged in the anteroposterior projection, the costal angle is obtuse, the ribs lie horizontally, and the intercostal spaces are expanded. The percussion note of the chest is loud and hollow (bandbox sound). The lower boundary of the lungs is considerably depressed, while the upper boundary is raised. The respiratory excursions of the lungs are limited; during inspiration the lower border of the lung moves down by 2-4 cm along the anterior axillary line, instead of by 8-10 cm normally. When the circumference of the thoracic cage is measured at the level of the 6th rib during inspiration and expiration the difference can hardly reach 1-2 cm. The lungs cover the area of absolute cardiac dullness. On auscultation of the lungs, one hears weakened respiration, prolonged expiration, and dry and moist rales, if emphysema is accompanied by bronchitis and pulmonary congestion. At spirometry, the vital capacity of the lungs is markedly reduced, the amount of air exhaled not exceeding 2,000 cc. The heart tones are muffled. There is an accentuation of the second sound over the pulmonary artery during a pressure increase in the pulmonary circulation. The pulse is quick and feeble. Pulmonary emphysema is known to develop by several phases. At the onset of the disease, the patient complains of a periodic cough and slight breathlessness on exertion. Later, laboured breathing occurs even on slight exertion and then at rest. In the final stage of pulmonary emphysema respiratory difficulty is joined by cardiac insufficiency due to hypertrophy and weakening of the right cardiac ventricle and relative insufficiency of the tricuspid valve with systolic pulsation of the liver, a positive venous pulse, and a systolic murmur at the xiphoid process; the latter entails a disturbance in the veins of the systemic circulation, accompanied with enlargement of the liver, ascites and peripheral oedema.

The *prognosis* in the initial stage of the disease is favourable if efficacious therapy of diseases responsible for respiratory insufficiency (bronchial asthma, bronchitis, pneumonia, etc.) is undertaken in good time, and the patient is provided with favourable working and living conditions.

Prophylaxis consists in prevention of various pulmonary diseases likely to cause respiratory failure, measures to protect patients against chilling and improve their housing and living conditions (because of the hazards of damp and overcrowded surroundings).

Treatment is symptomatic (cupping glasses, mustard plaster, expectorants, etc). Sulpha drugs and antibiotics are given for fever and pneumonia. Ephedrin is used to dilate the constricted lumen of the bronchi and bronchioles, and adonis and digitalis for symptoms of cardiovascular insufficiency; in severe cases euphyllin is administered intravenously (formula No. 27) and strophantine with glucose. In breathlessness and cyanosis, oxygen therapy is helpful (placing the patient into an oxygen tent for 20 minutes to 1-2 hours, or giving oxygen from a balloon—through a catheter inserted into the nose, or through a mouthpiece). For oedema diuretics are prescribed—hypothiaside (formula No. 46*a*) with potassium chloride (formula No. 54*a*), novurit (formula No. 43), phonurit (formula No. 43*a*) and other drugs.

LOBAR PNEUMONIA (PNEUMONIA CROUPOSA)

Lobar pneumonia is an acute infectious disease in which the inflammatory process invades the entire pulmonary lobe for which reason it is known as lobar as distinct from focal or lobular pneumonia, when the disease process is confined to one or several lobules. Lobar pneumonia was formerly regarded as an inflammation involving an entire lobe or several lobes. It has been found at the present time that the lungs are subdivided into segments (10 segments in the right lung, of which 3 are in the upper, 2 in the middle, and 5 in the lower lobe; and 8 segments in the left lung—four in each lobe). Correspondingly, an inflammation may extend both to the entire lobe and to separate segments. Lobar pneumonia is sometimes called fibrinous because in this disease the inflammatory exudate consists of fibrin.

Lobar pneumonia is characterized by a sudden onset, with affection of the entire lobe or one or several segments. Most commonly, the right lung is afflicted, primarily its lower lobe, possibly because the right bronchus is wider and shorter and deviates but slightly from the trachea; as a result the air is supplied to the lower lobes with utmost force. Lobar pneumonia runs a cyclic course.

Aetiology and pathogenesis. The causal organisms of lobar pneumonia may be pneumococci, streptococci, staphylococci, *E. coli* and sometimes Friedlaender's bacilli. Pneumococci were first discovered in 1818 in sections of the lung of a horse which had died of pneumonia. For their biologic properties they are subdivided into four types, the 1st and 2nd types being the most frequent cause of pneumonia and are encountered in healthy persons extremely seldom. The share of pneumococci, which were considered to be the principal cause of pneumonia has considerably reduced after a wide introduction of antibiotics and chemotherapy of infectious diseases, pneumonia in particular.

Pneumonia is contracted when pneumococci or other causal organisms have entered the lungs of a person susceptible to them. It has been established that introduction of a pneumococcus culture into the upper air passages of animals fails to induce pneumonia. If the same animals, however, have preliminarily been given an injection of a killed pneumococcus culture, i.e., sensitized, another injection of a live but already attenuated pneumococcus culture will induce pneumonia, particularly in case of a simultaneous injury to the nerves. The acute onset of lobar pneumonia, with a chill, high-grade fever, and sudden extension of the disease process to the entire lobe or a segment of the lung gives one grounds to regard it as a local manifestation of an allergic reaction. For lobar pneumonia to become firmly implanted, an important role is played by the condition of the mucous membrane of the upper air passages and by congestion in the lungs. At the same time the cilia of the ciliated epithelium lose their function as a barrier to infection and favourable conditions are thereby provided for pneumococci to gain entry into the lungs. Lobar pneumonia may also be contracted by the so-called haematogenous route by which pneumococci arrive from various regions with the blood stream, or by the lymphogenous route by extension of infection from bronchial lymph nodes. The disease may also be traumatic in origin and be consequent upon a contusion of the chest. Chilling and cold were also considered to be responsible for the occurrence of pneumonia as evidenced by its heightened rate in the cold season of the year, as well as by the development of pneumonia due to the influence of a sudden change in the air temperature and weather conditions. Cold and chill, however, may be regarded only as predisposing factors rather than the principal cause of lobar pneumonia.

Lobar pneumonia affects persons of any age but more commonly younger or elderly subjects. The predisposing factors are alcoholic and other intoxications, nervous and mental strain, physical overexertion, and chronic inflammations of the upper respiratory tract.

Pathologic anatomy. Pneumonia is known to run its course in the following four stages: (1) hyperaemia (congestion); (2) red hepatization; (3) grey hepatization; (4) resolution. The first stage is characterized by severe congestion of blood vessels and serous oedema of the affected lobe. The alveoli are dilated and contain air and serous exudate rich in fibrin. On section, the lungs are of a red colour. This stage lasts from 12 hours to 3 days. In the stage of red hepatization the alveoli contain a large number of erythrocytes while the air is displaced from them. They contain a considerable amount of plasma protein, fibrinogen, cells of the alveolar epithelium, and a small number of neutrophilic leucocytes. The effected lobe of the lung grows in volume two- or three-fold, the lung tissue becomes as dense as that of the liver. That is why this condition is known as *red hepatization*. This stage continues from 1 to 3 days. The stage of grey hepatization immediately follows that of red hepatization and makes its appearance on the 4th-6th day of the disease. Red hepatization turns to grey gradually and ununiformly. In this period, the leucocytes in the alveolar exudate increase in number and lend it a grey colour, while the lung tissue becomes extremely consolidated, with a pus-like mass discharged from it on pressure. This stage continues from 2 to 6 days. The stage of resolution begins from the 6th-7th day of the disease, and is characterized by proteolytic and autolytic processes. The fibrin becomes liquified and the leucocytes, pneumococci and desquamated alveolar epithelium undergo decomposition and dissolution. Only a small amount of exudate is expectorated in sputum form. Such a sequence of the progression of a pneumonic process in the lung has changed substantially after sulpha drugs and antibiotics have been introduced into clinical practice. A classical pathoanatomic picture of lobar pneumonia with a change of stages is still encountered in persons to whom sulpha drugs or antibiotic therapy are unavailable.

In the majority of cases, the lung exudate is quickly resorbed upon recovery, and in some patients it is retained or ceases to effuse. In the latter cases the exudate undergoes organization, is invaded by connective tissue and causes pulmonary cirrhosis (carnification) with a subsequent formation of areas of bronchiectasis. Sometimes the proteolytic or autolytic processes involve normal lung tissue and give rise to abscesses and gangrene. The attendant fibrinous pleurisy is also resorbed completely upon recovery or leaves thickened areas in the visceral pleura. In certain cases, croupous pneumonia is complicated by purulent pleurisy. The heart muscle is swollen, and a fatty degeneration and small inflammatory foci are encountered. The vessels of the pulmonary circulation also become affected because the right heart is forced to work in the presence of increased resistance in the pulmonary circulation caused by emptiness of the capillaries in the affected areas. Hypotonia comes on the scene, particularly when toxaemia is present. All these changes are very dangerous, especially for elderly and senile persons, since they are liable to bring about cardiovascular insufficiency, pulmonary oedema and collapse. Prior to the existence of the modern therapeutic methods, lobar pneumonia was often complicated by meningitis, most often serous, with an increase in the amount of cerebrospinal fluid and cerebrospinal pressure (up to 200 mm of water instead of 120 mm), and in the number of cells. Lobar pneumonia is accompanied by respiratory failure varying in severity with the number and size of the affected pulmonary lobes. Its appearance is associated with limitation of the respiratory surface of the lungs as well as with failure of venous blood to enter the inflamed lung tissue in the stage of hyperaemia and red hepatization to become oxygenated and relieved of carbon dioxide. Instead of the normal 95-96 per cent level of oxygen saturation of the blood this percentage is reduced to 80-93 per cent, which results in breathlessness and cyanosis of extremities. An extensive affection of the liver with marked symptoms of jaundice is a sign of bilious pneumonia. It should be taken into account, however, that in such cases jaundice is due not only to necrosis of the liver parenchyma but also to an intensified disintegration of erythrocytes whose haemoglobin is the source of the bile pigment bilirubin.

Of the biochemical changes discoverable by laboratory analysis attention is deserved by the increased nitrogen content of the blood and urine owing to an intensified disintegration of protein in the stages of grey hepatization and resolution, a reduction in the amount of chlorides, both in the blood and the urine, and return to normal soon upon recovery. At the peak of the disease the sugar content of the blood is increased, and during the glucose tolerance test (50 g) the sugar curve becomes diabetic in character, failing to return to the initial figures within 2-3 hours of the test.

Clinical picture. The incubation period is undeterminable, save for traumatic lobar pneumonia which occurs within several hours of injury. The disease has an acute onset, with chill and a temperature rise to 39-40°C, headaches, sometimes vomiting, pains in the chest. The pain corresponds in location to the affected area and most often is felt in the lower parts of the thoracic cage, extending along the anterior axillary line, and being confined to a very limited region. It becomes worse on pressure, coughing, and sneezing, and irradiates to the back, arm, hypochondriac region, abdomen and the thigh. The patient assumes a forced posture, lying on his back with a raised trunk since he cannot lie on the affected side for pleural pains. At the peak of the disease, the patient prefers to lie on the unaffected side, which facilitates sputum discharge from the diseased lung as well as sputum expectoration. With a deep-seated pneumonic focus (central pneumonia) as well as in affection of the upper lobe painful sensations may be totally absent. Sometimes, they may be confined to the right iliac region and be mistaken for appendicitis.

The patient's face is cyanotic, particularly the lips, the skin is reddened and slightly damp. The cheeks are flushed, mainly on the affected side. The lips, wings of the nose, and cheeks are covered with herpetic eruptions (herpes labialis, nasalis, buccalis).

Fever is permanent and persists at a high level during 4-6 days. The temperature drops to normal critically within 12-24 hours, with perspiration, debility, hypotonia, and a quick and feeble pulse. A lytic decline of body temperature during several days is seen mainly in subjects weakened by emaciating precursory diseases. Sometimes, fever again climbs up before a drop (precritical elevation). In some cases, 2-3 days after the temperature declines to normal it again becomes subfebrile, possibly because of resorption of the remnants of exudate. Sometimes, a drop in temperature is again followed by a rise, which is the so-called pseudocrisis (false crisis). The wide application of sulpha drugs, and especially antibiotics, has radically changed the course of pneumonia, in particular the character of the attending fever, which often decreases to the normal body temperature within 48-72 hours.

Cough is one of the permanent symptoms of lobar pneumonia, occurring together with pain in the chest and may be absent only in elderly and debilitated persons. In the early days of the disease the cough is excruciating.

Sputum which is at first viscid and vitreous, becomes rusty in colour on the 2nd day as a result of disintegration of the haemoglobin of the erythrocytes; it is sometimes blood-streaked and in certain cases (in particular in mitral disease and congestion in the pulmonary circulation), blood-stained. Sputum is expectorated with difficulty and sticks to the teeth and lips; it contains numerous erythrocytes, leucocytes and threads of fibrin. In the period of resolution of the lung process sputum turns mucopurulent, and is easily expectorated.

Changes in the respiratory organs. Examination of the thoracic cage reveals a delay in the respiratory movements on the affected side. The affected lobe grows in size while the intercostal spaces become flattened. The breathing is quickened to 30-40 respirations per minute, and is shallow because of sharp pains on the affected side. In the hyperaemic stage a dulled tympanitic sound is elicited on percussion over the affected area, on auscultation crepitation (crepitatio indux) is heard at maximal inspiration. The vocal fremitus and bronchophony are altered but slightly. In the stage of red and grey hepatization absolute dullness is heard on percussion over the affected region where one can also distinctly hear bronchial respiration, intensified vocal fremitus and bronchophony, if the bronchus is not occluded with viscid secretions, while crepitation is heard no longer. In concomitant bronchitis dry rales are heard. During the resolving stage of the disease, i.e., in the period of exudate resorption, a dulled tympanitic sound is heard, bronchial respiration becomes uncertain, and crepitation (crepitatio redux) reappears on maximal inspiration.

Changes in the cardiovascular system. A patient with lobar pneumonia develops cyanosis of the hands, face, and mucous membranes, resulting from insufficient oxygenation of the blood. The latter condition is brought about by a disturbance of normal gas exchange in the lungs, impairment of the function of the right cardiac ventricle, as well as by a toxic affection of the myocardium and the capillary network.

Circulatory insufficiency is the most dangerous symptom in lobar pneumonia. A rise in body temperature by 2° entails an increase in basal metabolism by 15 per cent. This intensifies oxygen consumption which is ensured by an increase in the cardiac output or by an increased release of arterial blood oxygen by the capillaries. An increased work of the heart in lobar pneumonia is also due to an obstacle to the blood flow in the pulmonary circuit, which entails an icrease in load on the right cardiac ventricle. An increased resistance in the pulmonary blood flow is manifested by intensification of the second tone over the pulmonary artery. In consequence of the above-mentioned causes, as well as toxaemia the myocardium undergoes fatty and hyaline degeneration. The pulse rate fluctuates between 90 and 100 beats per minute. A quickened pulse rate of over 100 beats per minute is an ominous sign. Cardiac arrhythmia (extrasystole, sometimes auricular flutter, auricular fibrillation known as delirium cordis) is often noted. The arterial pressure is reduced. As the right cardiac ventricle weakens, the formerly accentuated second sound over the pulmonary artery disappears, the right ventricle and the liver grow in size, the veins on the neck become swollen and severe cyanosis develops. Cardiovascular failure may come on in any period of the disease, but is particularly frequent during a critical decline of body temperature. In such cases, the pulse becomes small and feeble, and cold perspiration, a pale face, and a reduction of the arterial and venous blood pressure occur, i.e., the patient suffers a collapse.

Changes in the organs of the abdominal cavity. There is a complete or partial loss of the appetite, and vomiting, intense thirst, meteorism and predisposition to constipations. The tongue has a white coating. Toxic hepatitis with jaundice sometimes develop.

Condition of the urinary organs. From the first day the amount of urine is markedly reduced. The urine is of an intense red colour and a high specific gravity. It contains a large number of nitrous substances—urea and uric acid formed as a result of an intensified cell decomposition in the body, and common salt in trace quantities. Very often albuminuria and cylindruria develop because of fever. Haematuria is evidence of the development of focal nephrosis. In toxic hepatitis bile pigments and urobilin appear in the urine. In the early days after the crisis there is intensified diuresis and discharge of retained common salt with the urine.

Nervous system. In certain cases, the patient's conscience is dimmed or confused. The patient is sometimes excited. This is particularly frequent in alcoholics and patients with a history of mental disease. They jump out of bed and try to escape, often through a window. This state of anxiety sometimes occurs after a crisis also.

Blood. In lobar pneumonia the blood undergoes very characteristic changes. These are as follows: neutrophilic leucocytosis with an increase in the number of rod neutrophils, a reduction in the absolute and relative number of lymphocytes and an increase in the number of monocytes. With the modern methods of therapy, the number of esosinophils is preserved, and sometimes becomes even larger. A high-degree leucocytosis is a favourable sign; with an excessive increase in the number of leucocytes, however, the prognosis is worse. Severe forms of pneumonia often run their course without marked leucocytosis, even with leucopenia. One of the signs pointing to a severe course is a distinct toxic granulation of neutrophils, when the degeneration index (proportion of degenerative neutrophils to normal) is higher than 50 per cent. The total protein content of the blood serum is reduced with a simultaneous decrease in the level of albumins and an increase in globulins. There is a growth in the amount of fibrinogen, a reduction in the number of thrombocytes, an elevation of the erythrocyte sedimentation rate (ESR), and in grave cases an inverse relationship is noted between the number of leucocytes and the ESR, i.e., when the former is reduced, the latter is accelerated. The content of chlorides in the blood is lessened since they are evidently retained in the tissues.

X-ray picture. In the majority of cases of pneumonia a uniform and diffuse shadow with no well-defined boundaries and thin like a weil is noted in the period of hyperaemia. In the period of hepatization, there is an intense shadowing of a part or the whole of a lobe and sometimes of several lobes.

Variants of the course of lobar pneumonia. In addition to the above-described typical form, in certain cases atypical forms of lobar pneumonia are encountered.

Atypical forms are found in debilitated persons; they start without a chill and run their course with a slightly elevated temperature. The local symptoms in the lungs are mild, but the general condition is extremely grave.

Pneumonia of the upper lobe usually runs a severe course, with considerable changes in the nervous system.

Central pneumonia is characterized by the localization of the disease focus in the perihilar zone without extension to the lung periphery. The disease has an ordinary onset, with fever and chill, sputum discharge of a rusty colour, and leucocytosis. Characteristic percussion notes and auscultation sounds are absent because of the central localization of the inflammatory focus. The diagnosis rests on the presence of a rusty sputum discharge, an intense neutrophilic leucocytosis, and x-ray findings.

Migratory pneumonia. The pneumonic process may spread from one lobe to another and be attended with a repeated rise in temperature. In fatal cases, autopsy reveals foci in the stage of hyperaemia in addition to areas of hepatization. Migratory pneumonia often affects old persons and those wasted by prolonged diseases.

Complications of lobar pneumonia. These are serofibrinous wet pleurisy, purulent pleurisy, pulmonary abscess and gangrene, and pericarditis. The exudate may collect in the lower portions of the pleural sac or in the interlobar fissures. The exudate is readily resorbed with the termination of pneumonia, though the temperature returns to normal after a certain delay. The supervention of purulent pleurisy, usually when the disease is drawing to a close, complicates its course. The temperature which declined earlier again climbs up and becomes hectic, with considerable neutrophilic leucocytosis and intense pains on the affected side.

In some cases, the resolution of pneumonia is delayed, and the exudate undergoes organization, i.e., connective tissue begins to form in its place. This leads to a later appearance of areas of bronchiectasis. Sometimes, the process of exudate resorption in the alveoli extends to the lung parenchyma, in which case an abscess and less often gangrene of the lung are provoked.

Serous and purulent meningitis were formerly fairly frequent diseases. In serous meningitis there is mild rigidity of the occipital muscles and a slight Kernig's sign. Lumbar puncture reveals an increased pressure in the spinal canal (200 mm instead of 120-150 mm of water), an increase in cell number (more than 10 cells to 1 ml of fluid), an insignificant increase in the amount of protein. Purulent meningitis is a very rare disease.

Differential diagnosis of lobar pneumonia should be made between wet pleurisy, bronchopneumonia, pulmonary infarction, caseous (cheesy) pneumonia, acute appendicitis, and cholecystitis.

In wet pleurisy the area of dullness is situated above the lower parts of the thorax and its upper border has a highly characteristic outline (Damoiseau's curve, Rauchfuss-Grocco's triangle). The dullness is markedly audible with a femoral resonance. The mediastinum, the heart in particular, is displaced to the healthy side. In the area of dullness, vocal fremitus and respiratory murmur are absent; above the area of dullness, however, vocal fremitus and bronchophony are intensified. In sinistral (left-side) pleurisy Traube's semilunar space is filled up. The fever is not as high as in pneumonia, although it persists for a long time. A fluid is obtained on puncture of the pleura.

Bronchopneumonia runs its course without any characteristic change of stages, neither has it any percussion and auscultation sounds characteristic of lobar pneumonia.

It is not always easy to differentiate lobar pneumonia from pulmonary infarction. The latter disease, just as lobar pneumonia, begins with a chest pain and blood-spitting. In lobar pneumonia, however, these symptoms are preceded by a high-grade fever. X-ray findings are not sufficient evidence for ruling out the possibility of lobar pneumonia. In pulmonary infarction, streaks of pure blood are found in the sputum, whereas in pneumonia the sputum discharge is blood-stained. In the former disease, the affection is smaller in area, pyramidal in shape, and does not correspond to the lung boundaries. The diagnosis of pulmonary infarction can be established with certainty in the presence of sources of emboli in the veins of the general circulation, the right ventricle, pelvic and haemorrhoidal veins, etc.

The greatest difficulties are encountered in a differential diagnosis between lobar pneumonia and caseous tuberculous pneumonia, especially in the early days of the disease, when no *Mycobacterium tuberculosis* is present in the sputum. In patients with caseous pneumonia. the temperature rises slowly, the face is usually pale, and the disease process is confined mainly to the upper lobes. As distinct from lobar pneumonia, caseous pneumonia runs its course with no stages and for a long time. Highly valuable for diagnosis are x-ray findings in the lungs, as well as examination of sputum, which at the end of the disease may be found to contain the causal organisms of tuberculosis.

Prognosis. The death rate for lobar pneumonia before the advent of sulpha drugs and antibiotic therapy ranged from 15 to 25 per cent. Today, the disease is rarely fatal.

LOBULAR PNEUMONIA OR BRONCHOPNEUMONIA (PNEUMONIA LOBULARIS)

Lobular pneumonia is the collective term for inflammatory processes of variable aetiology, occurring in the lungs and confined to one lobule or a group of lobules. Most commonly, lobular pneumonia follows bronchitis as a result of infection spreading from the bronchi to the lung tissue, for which reason lobular pneumonia is also called bronchopneumonia. Cases are frequent, however, of inflammatory processes emerging in the lobules by way of the blood stream. That is why pneumonia localized in separate lobules or a group of lobules, with mucous or mucopurulent matter, contained in the alveoli, should with better reason be called lobular pneumonia. This disease is found more often than lobar pneumonia, particularly in elderly and senile persons.

Aetiology and pathogenesis. Lobular pneumonia is secondary to bronchitis and occurs in various infectious diseases (measles, whooping cough, influenza, typhoid fever), diseases of the kidneys, circulatory disturbances, gas poisoning (with phosgene, etc.), on inspiration of infective or toxic material during general anaesthesia (aspiration pneumonia), due to atelectasis (collapse) of the lungs, stasis (hypostasis in the lungs), and in bed-ridden patients. Lobular pneumonia may also occur due to the entrance of infection into the lungs with the blood stream (metastatic pneumonia).

The causative organisms of lobular pneumonia are pneumococci of groups III and X, streptococci, staphylococci, and influenza virus. The causal agents of plague and anthrax are also capable of producing pneumonia, though of a specific character.

The infection enters the lungs by way of the bronchi, the blood stream, and the lymphatic vessels. During inspiration, microorganisms enter the bronchi with the air inhaled. With a normal excitability of the bronchial mucosa, the cilia of the epithelial layer capture them, so they are expelled by a cough or are ingested by phagocytes. During inflammatory processes, as well as in total and partial vitamin A deficiency, with atrophy of the epithelium lining the airways, the cilia lose their function as a barrier to the entry of microorganisms. The latter settle on the bronchial walls and cause their inflammation with a subsequent spread of the disease process to the pulmonary parenchyma. Lobular pneumonia often results from various foreign matter, such as food particles, vomit, pus, blood, mucus, etc., being inspired into the bronchi with reduced reflex excitability. This is found in patients with cerebral haemorrhage, injuries to the chest, face, or jaw, in alcoholic intoxication, during ether anaesthesia, uraemia, paralysis of the muscles of deglutition, etc. In such cases, the development of pneumonia is provoked by obstruction of the bronchi with foreign matter and emergence of pulmonary atelectasis. Air is absorbed from the occluded areas, so that favourable conditions are provided for an inflammatory process. Analogous conditions for the occurrence of pneumonia arise in patients confined to bed for a long time, i.e., debilitated persons, those with derangements in the pulmonary circulation, bronchiectasis, and bronchial asthma, when inadequate ventilation of the lungs contributes to the development of atelectasis. Lobular pneumonia may also be haematogenous in origin, i.e., result from the entry of infection with the blood stream into the peribronchial tissue, bronchi and alveoli. Haematogenous pneumonia is encountered in patients with septic diseases, infected wounds, operations performed on the abdominal cavity, in particular in the presence of an infective focus in peptic ulcer, also with diseases of the uterus and other disorders, in which pathogenic organisms may find their way into the lungs through the blood stream and are likely to cause metastatic pneumonitis.

Pathologic anatomy. Foci of inflammation are commonly confined to the lower lobes in one lung or both, and vary in number and developmental stage. As a result of this, the lungs show a marble pattern on section (acquiring a motley pattern varying in hue from red to grey-yellow) and are slightly prominent above the surface. Around the foci of pneumonia one finds emphysematous swollen areas of pale-coloured lung tissue, with sunken blue-red areas of atelectasis visible between the inflamed areas. In certain cases, the inflamed lobules coalesce to give a false impression of affection of the entire lobe. In the inflamed sections, the alveoli contain liquid exudate of a haemorrhagic, purulent, putrid, but most commonly serous character. The exudate contains a small amount of fibrin, cells of the alveolar and bronchial epithelium as well as leucocytes and erythrocytes. Putrid decomposition is most often found in patients with aspiration pneumonia, haemorrhagic exudate in patients with influenzal pneumonia.

Clinical picture. Lobular pneumonia is characterized by fever, coughing, pain in the chest, and reaction of the haemopoietic system.

The disease develops gradually, and most commonly pneumonia is preceded by bronchitis. The supervention of pneumonia is found from the appearance of signs of toxaemia, as manifested by lassitude, debility, headache, and a rise in body temperature. Fever runs an irregular course, now climbing up, now going down. These temperature variations are caused by abatement of morbid symptoms in some of the foci and appearance of new foci of inflammation in the pulmonary tissue. Fever persists from several days to 1-2 weeks and declines gradually. In debilitated and old persons, pneumonia sometimes runs its course without a marked temperature change and sometimes without any fever at all. The patient's breathing is rapid and shallow, with 30-40 respirations per minute. There is breathlessness and cyanosis of the face, depending on the extension of the inflammatory process and associated with the accompanying bronchitis, reduction in the respiratory surface and occlusion of the lumina of numerous bronchioles and alveoli. Important signs of pneumonia are pains in the chest, especially on deep inspiration, caused by attendant pleurisy, and cough, commonly with a mucopurulent easily expectorated sputum. The pulse rate is quickened, the arterial pressure is reduced, while the condition of the cardiovascular system depends on the spread of the disease process. A very characteristic sign of lobular pneumonia is neutrophilic leucocytosis within a range of 10,000-15,000 per cu mm of blood and an accelerated ESR, while at the peak of the disease no eosinophils are detected in the blood. The level of fibrinogen in the blood is usually normal or only slightly elevated. The patient, when not unconscious, assumes a semireclining position.

The findings of physical examination depend on the depth of localization and size of the focus. A delay in the respiratory movements of the thoracic cage on the affected side is noted in pleural involvement. Only in the presence of large pneumonic foci located closer to the periphery can one determine dullness of the percussion note and the presence of harsh respiration of a bronchial resonance, an intensified vocal fremitus with bronchophony as well as small and medium bubbling rales and crepitation. Dry rales caused by diffuse bronchitis are heard all over the lung. In coalesced lobular pneumonia the percussion note is dulled. If the disease process is localized centrally or occurs in a debilitated patient with shallow respiration, neither percussion nor auscultation may be helpful in detecting bronchopneumonia. It should be noted that quite different findings are obtained on percussion and auscultation of the lungs, since foci of pneumonia at different stages of development exist in the lungs simultaneously. For example, foci in the stage of hyperaemia and resolution give out a tympanitic sound on percussion and crepitation on auscultation, and dullness and bronchial respiration in foci of consolidation respectively. This variety of physical signs is a distinctive feature of lobular pneumonia as compared with the lobar form.

The liver and spleen are not enlarged. The digestive organs show no characteristic signs, save for the tongue which has a white coating. The urine is found to contain a small amount of protein and erythrocytes. X-ray examination of the lungs reveals multiple foci of a variable size, irregular form and different intensity. Foci localized centrally do not always give a distinct shadow. Shadowing is usually markedly intense at the root of the lung owing to enlargement of the lymphatic glands, and sometimes because of inflammatory foci lying proximally to the lung hilum. Multiple foci of pneumonia sometimes produce a picture resembling that of haematogenous, even miliary tuberculosis. In the resolution stage, the major part of the exudate is resorbed through the lymphatic channels and only an insignificant amount is expectorated with sputum.

Course and complications of the disease are variable. In some cases, it ends in recovery within a few days, and sometimes lasts for weeks. In persons debilitated by a grave infection and in old age pneumonia runs a torpid course. Among its complications are lung abscess, pulmonary gangrene, and pneumosclerosis. In certain cases, the exudate is invaded by connective tissue, so that the pneumonic focus acquires the consistency of flesh, i.e., undergoes carnification (from Latin *caro*—flesh). In these sections areas of bronchiectasis may form later. Measles and whooping cough pneumonia is liable to activate pulmonary tuberculosis present in a latent state.

Severe and lingering forms of lobular pneumonia must be differentiated from pulmonary tuberculosis and wet pleurisy.

INFLUENZAL PNEUMONIA (PNEUMONIA GRIPPOSA)

Influenzal pneumonia is a focal pulmonary inflammation complicating the course of influenza. During an epidemic of influenza the occurrence of pneumonia is sharply increased. Pneumonia found in patients with influenza varies in origin. At present quite a few cases are known of lobular pneumonia caused by influenza virus without any other pathogenic agents, pneumococci in particular.

The body of an influenza patient with a reduced resistance is a favourable environment for the reproduction of various microorganisms, not only pneumococci but also *Staphylococci aureus*, *Streptococci viridans*, and *E. coli*. These microorganisms may produce pneumonia; their proportion has noticeably increased after the introduction of chemotherapy and antibiotics into medical practice.

Influenzal virus pneumonia is an independent disease. When it is joined by a bacterial infection it tends to change its clinical course. In the development of influenzal pneumonia a role is played by the direct action of virus on the lung tissue and vessels. Later, bacterial flora may develop in the affected areas of the lungs and give rise to virus-bacterial pneumonia. From this point of view, the following classification of the types of influenzal pneumonia may be useful: (1) virus, (2) virus-bacterial, and (3) bacterial.

Pathologic anatomy. The patient is found to have acute catarrhal and haemorrhagic tracheobronchitis, bronchiolitis with ulcerations, and peribronchitis. The lungs show serous, serohaemorrhagic, often haemorrhagic foci of pneumonia with predisposition toward abscess formation. Wet pleurisy also develops frequently.

Clinical picture. Virus and virus-bacterial pneumonias develop slowly, sometimes acutely, with fever rising to 39-40°C, often with chills and symptoms of general toxaemia: headache, dull pains throughout the body, adynamia (prostration), and lassitude. In the early days of the disease, the patient suffers from coryza (running nose), cough, at first dry, later with a mucous sputum discharge, chest pain and breathlessness. In haemorrhagic influenzal pneumonia a bloody sputum is discharged since the first day of the disease, and the rate of breathing is 40-50 respirations per minute. Changes revealed on percussion and auscultation depend on the size of the affected area. The percussion note is dull, with harsh respiration and dry and moist rales. These signs are changeable and inconstant, while bronchial respiration and crepitation are heard very seldom. The heart boundaries are expanded, with muffled tones and systolic murmur over the apex. The number of erythrocytes in the blood is increased in respiratory insufficiency (compensatory polycythaemia).

Other symptoms are neutrophilic leucocytosis, as well as leucopenia, eosinopenia, and an increase in the number of monocytes. X-ray examination reveals an expansion of the lung shadow (at the roots), especially on the affected side, a sharp enlargement of the lung pattern and deformation caused by excess blood in the lung vessels.

Course. The onset of pneumonia coincides with influenza. In other cases, a fall in body temperature in a patient with influenza is followed by a new rise, with symptoms of toxaemia typical of pneumonia. At the same time, changes in percussion and auscultation sounds over the lungs are notable. This is late pneumonia, which for its clinical course does not differ from early pneumonia. The most severe course is run by haemorrhagic pneumonia: with acute haemorrhagic pulmonary oedema, general cyanosis, hypotonia, a serosanguineous sputum discharge, and a severe general toxaemia.

Influenzal lobular pneumonia usually resolves within 11 days. Sometimes, subfebrile temperature persists for several weeks. In such cases, pneumonia takes a torpid course and often leaves scar tissue in the lungs (carnification, areas of bronchiectasis, etc.).

Complications. Among the complications the most frequent are dry and wet pleurisy (serous, serofibrinous, seropurulent), an activation of tuberculosis, paranasal sinusitis, bronchiectasis, and, though very rarely, meningoencephalitis.

Traumatic pneumonia occurs fairly often, particularly in persons with injuries to the head (25 per cent of cases), the thorax (15 per cent), the abdomen (12 per cent), the upper extremities (6 per cent), and the maxillofacial region and in traumatic sepsis. Traumatic pneumonia sometimes results from blood congestion in the lungs, particularly in the lower lobes.

Pneumonia in cranial trauma is seen in the early hours and days of injury. In this condition, the physical signs are quite unmarked, with slightly weaker respiratory sounds and moist rales in the affected area. The temperature is either normal or slightly elevated.

Pneumonia in maxillofacial injuries is acute in onset, with a high-grade fever, leucocytosis, and productive cough. There is a dulled percussion note over a large area as a result of coalescence of inflammatory foci. Bronchial respiration and later moist rales are heard. Traumatic pneumonia is often complicated by pulmonary suppurations.

Pneumonia in thoracic injuries is identified with difficulty because the collection of blood in the pleural cavity (haemothorax) interferes with the physical and x-ray examination of the inflammatory focus. In such cases, the diagnosis of pneumonia is usually facilitated by the presence of indirect symptoms, such as coughing with sputum discharge, sometimes blood-stained, leucocytosis, an accelerated ESR, etc.

TREATMENT, PROPHYLAXIS AND CARE OF THE PATIENT IN LOBAR AND LOBULAR PNEUMONIA

A patient with pneumonia ought to be accommodated in a well-ventilated and illuminated ward with windows on the sunny side. The head of the bed should be raised a little so as to aid the patient's breathing. To prevent complications (pulmonary gangrene, stomatitis, parotitis, etc.), the tongue and the mucous membrane of the oral cavity must be wiped with tampons soaked in 2 per cent hydrogen peroxide. Herpetiform blebs should be painted with zinc ointment. A cleansing enema must be used for constipations. In flatulence of the intestine it is recommended to insert a gas-release tube. In case of excitement and severe headache the patient's body should be wiped with a wet sponge, and ice applied to the head. In such cases, a round-the-clock watch is maintained at the bedside.

Of the pharmaceutic remedies it is helpful to use sulpha drugs (sulphazole, norsulphazol, sulfadimezine, etc.), according to the following scheme: on the 1st day a 2 g dose of the drug is given immediately, followed by 1 g doses every four hours; on the 2nd day 1 g doses are given every four hours; on the 3rd, 4th and 5th day, 4 g are given, i.e., 1 g every six hours. A total of 25 g is administered throughout a course of treatment in uncomplicated pneumonia.

To ensure a better secretion of urine and prevent the formation of renal calculi from drug crystals, one is advised to wash down a sulpha drug with soda water (2-3 g of sodium bicarbonate to a glass of water) or mineral water because no sulphanilamide calculi are known to form in alkaline water. It is prohibited to use sulphate purgatives during sulpha drug therapy. In addition to sulpha drugs, antibiotics (penicillin, streptomycin, biomycin, tetracycline, terramycin, etc.), which arrest the growth of pneumococci, are also used on a wide scale. Antibiotics are especially helpful in intolerance of sulpha drugs, for conditions caused by sulpha-resistant pneumococci as well as in cholelithiasis, nephrolithiasis, nephritis, hepatitis, anaemia and leucopenia.

Penicillin and streptomycin are administered intramuscularly in fractional doses, penicillin in doses of 100,000 U in a 0.25 per cent novocain solution 6-8 times daily, and streptomycin in doses of 500,000 U twice daily during a period of 4-5 days. Biomycin or terramycin in daily doses of 100,000-150,000 U can be used in tablet form 4-6 times daily. Therapy with sulpha drugs and antibiotics is known to be followed sometimes by an allergic reaction such as dermatitis or fungal diseases (candidiasis).

In addition to sulpha drugs and antibiotics, patients must be given bromide and luminal which have a soothing effect on the nervous system and improve sleep. The cardiovascular system is toned up by means of caffeine and camphor, and in collapse with strychnine, strophanthin, sympathol. Cough and pain are relieved by the applications of codeine, dry cupping glasses, and mustard plasters. Thermopsis and ipecacuanha are recommended as expectorants. In the case of a slow resolution of the infiltration physiotherapy is prescribed, such as applications of diathermy and Sollux light to the affected area. Oxygen therapy is extremely helpful. The patient's food must be liquid, readily digestible, highly nourishing and rich in various vitamins (dry crusts, milk, kefir, fruit jelly, milk cream, soft-boiled eggs, fruit juices, gruels, etc.). After a return of body temperature to normal, each patient feeling well should still be confined to bed for another 10 days, after which time he may be discharged from hospital. Work should not be resumed earlier than 3-4 weeks after a stable decline of temperature. To prevent the risk of pneumonia one will do wise to avoid crowded surroundings, chilling, and abuse of alcohol. Sports and physical culture contribute effectively to the hardening of the body.

Lobular pneumonia may be warded off by efficient care and treatment of the precursory disease. Aspiration pneumonia is prevented by means of gargling, wiping the oral mucosa and tongue with a gauze tampon as well as by displaying caution when feeding the patient. To forestall the occurrence of hypostatic pneumonia in a patient restricted to bed for a long time, he should be turned from side to side and whenever possible helped to sit up in bed. Respiratory gymnastics, which considerably improve the blood circulation in the lungs are also recommended. Atelectatic pneumonia may be prevented by measures taken against the risk of aspiration of foreign bodies, by clearing one's throat forcibly which is very important in cases of penetrating thoracic wounds when the effusing blood and mucus obstruct the bronchioles and cause atelectasis.

NONBACTERIAL PNEUMONIA

Psittacosis

Psittacosis is a disease rare in man. It is transmitted to human beings from parrots and other birds. It is supposed, however, that there exists psittacosis virus transmitted from person to person without the participation of birds. The largest epidemics of psittacosis invading most of the countries of Europe and Asia were reported in 1929-30.

Aetiology. The causal organism of psittacosis is a filterable virus which is discovered in the blood of sufferers within five days of onset and in the urine after the 20th day of the disease. Virus infection is harboured in parrots, sick persons or carriers of latent infection. In birds, the virus is excreted with faeces and nasal mucus. Their plumage may be soiled with faeces (during flux), which dry up into dust particles and become airborne. The latter are carried far and wide by the wind and are likely to infect persons who have no close contact with birds. The infection is transmitted on airborne droplets and through a bite, especially when a bird is being fed through the mouth. The most susceptible to psittacosis are persons above 50 years of age and the least susceptible are children under 10 years. In the cold season of the year, there is a certain increase in psittacosis incidence. The virus is isolated by intraperitoneal infection of albino mice and cultivation of microorganisms on chicken broth.

Pathologic anatomy. Inflammatory lesions are confined to the lungs, throat and trachea. The spleen is enlarged. The liver and kidneys show changes in the parenchyma, the lungs undergo tissue consolidation, while the alveoli are occluded with fibrin, erythrocytes, and leucocytes. The bronchial mucosa is desquamated.

Clinical picture. The disease is of a sudden onset, with fever rising to 39-40°C, severe headache, lumbar pains, and pain in the legs. The appetite is reduced or absent altogether, the tongue is coated, and nausea and vomiting occur sometimes. The patient complains of insomnia. His mood changes alternately from excitement to dejection. On the 4th day of the disease there is a dry cough (which becomes moist only after a temperature drop). Beginning from the same day, signs of pneumonia are noted as manifested by delicate crepitating or small and medium bubbling rales. Later, the percussion note is dulled. Most commonly, the disease process is confined to the lower lobe of the left lung. X-ray examination of the lungs shows a uniform shadow covering one or several lobes. Pleurisy is a rare occurrence. The heart sounds are muffled, the blood shows the presence of leucopenia, and in the period of recovery leucocytosis with lymphocytosis. The ESR is accelerated not only when the disease is at its height, but also in the period of convalescence. Complete resorption of the infiltrate does not occur earlier than the 3rd-4th week of the disease. Relapses of psittacosis run a short course lasting 5-7 days. Psittacosis occurs either in a mild ambulant or a severe form. Among its complications, bronchitis and pleurisy are the most frequent. For its clinical course, psittacosis resembles typhoid, leptospirosis, influenza, malaria, and pulmonary tuberculosis. The disease confers immunity.

The *diagnosis* is verified by isolating virus from sputum and blood, by a reaction of complement fixation with the patient's serum, as well as by an intracutaneous test.

Treatment and prophylaxis. Treatment is with biomycin (aureomycin), terramycin, and penicillin, which abate fever and eliminate toxaemia in 48-72 hours. Prophylaxis consists in examination of birds for infection with psittacosis virus and their destruction, if necessary. All objects with which sick birds have been in contact are subject to disinfection with liquid solutions.

Ornithosis

Ornithosis is an acute infectious (viral) disease transmitted to man from sick birds, usually from virus carriers with a latent form of infection. The most common carriers of ornithosis are pigeons, turkeys, chickens, pheasants, and, among the wild species, harbingers, white American herons, silvery sea-gulls, orioles, and wild ducks. Ornithosis may also be contracted from domesticated mammalian animals (cows, cats, etc.). The disease occurs as occasional cases or familial outbreaks (in dove-breeders). A large number of persons may contract ornithosis simultaneously during a mass slaughter of ducks, air pollution with down when birds are being plucked. The ornithosis virus is related to that of psittacosis. For their pathologic anatomy the diseases are identical.

Clinical picture. The incubation period lasts from 6 to 20 days. The prodromal period continues from 2 to 3 days, and manifests itself by debility, nausea and apathy. The onset of the disease is characterized by headache and acute lumbar pain. Pneumonitis appears from the 3rd-4th day, with a shorter percussion note over the affected area and crepitant rales. Pneumonia is usually unilateral. The patient complains of full loss of appetite and constipation. The tongue is coated, with a red rim and imprints of teeth along the margins. The liver is enlarged and tender on palpation. The spleen can be felt with the fingers, though not always. Among the cardiovascular symptoms, dullness of heart sounds, hypotonia and bradycardia are notable. The patient complains of a headache and insomnia. He is periodically excited, at times is prone to weep, and languid. The red blood count is unchanged; there is leucopenia with a leftward shift, aneosinophilia, and accelerated ESR. X-ray findings are identical with those in psittacosis. Exudate resorption is slow, and is completed when the disease has been in progress for a month.

The course of the disease may be mild, with a subfebrile body temperature, terminating in recovery within 2-3 days. Severe forms run their course with a high fever, and sometimes have a lethal outcome. Relapses, just as in psittacosis, occur in the 3rd week of the disease, and take a mild and brief course. The immunity conferred by ornithosis is less stable than that acquired in psittacosis.

Treatment and prophylaxis are the same as those for psittacosis.

Eosinophilic Pneumonia

Eosinophilic pneumonia is an allergic disease accompanied with formation of infiltrates in one or both lungs and high eosinophilia. Infiltrations are also observed in other organs: the liver, stomach, pancreas, etc. This has prompted many authors to give eosinophilic pneumonia other names, such as acute febrile eosinophilic asthma, benign eosinophilic leukaemia, infiltrative eosinophilia, etc.

Aetiology. Eosinophilic pneumonia is found with those forms of helminthic infestations when the larvae pass through the lungs (ascarides, *Necator americanus* hookworms, etc.). Cases have been reported in the literature of a massive outbreak of eosinophilic infiltration of the lungs at a sanatorium in Switzerland after eating fresh lettuce contaminated with *Ascaris* larvae. Eosinophilic pneumonia may also be due to the irritant action of allergens of vegetable origin (pollen, lily-of-the-valley). Cases have been described of eosinophilic pneumonia in personnel of grain storages, flour mills, bakeries, while other cases have been traced to the administration of sulphonamides, PASA, antibiotics (penicillin, streptomycin), urethane, barbiturates, a contrast substance for x-ray examination, salvarsan, etc. Eosinophilic pneumonia is found in patients with bronchial asthma during an attack and persists for several weeks after the process has been aborted. This type of pneumonia does not respond to antibiotic therapy, while failure of attempts to find bacteria in sputum cultures is evidence of its nonbacterial origin.

Pathologic anatomy. The bronchial walls are oedematous, and infiltrated by eosinophils, while their lumina are occluded with exudate. The alveoli also contain eosinophils. Eosinophilic infiltrations are present in the lobules, as well as in the liver, the prostate, the uterine appendages, and the pancreas. All this points to a haematogenous extension of the disease process, i.e., with the blood stream.

Clinical picture. The typical patient has a history of allergic diseases (urticaria, eczema, migraine, etc.). The disease often develops under the influence of allergens of vegetable origin. In many cases, eosinophilic pneumonia is preceded by catarrh of the upper air passages. The disease is abrupt or gradual in onset, and is prone to relapse. In the case of a gradual onset the patient complains of debility, early fatigue on slight exertion, general malaise, breathlessness, cough with scanty sputum discharge and a low-grade fever. On objective examination a small number of dry and moist rales are found in the lungs. In the majority of cases, eosinophilic pneumonia is detected accidentally. An x-ray examination of the lungs shows shadows of a variable size, localization and intensity, which are apt to a relatively rapid reversal. The blood is found to have excess leucocytes with an increase in the number of eosinophils to 20-80 per cent (mature eosinophils). The ESR is moderately accelerated. The sputum contains mature eosinophils, and is blood-streaked when expectorated in agonizing cough, but contains no pathogenic bacteria. The x-ray of the lungs reveals infiltrations, now isolated, now multiple, which are localized in different areas of one or both lungs.

The disease does not last longer than a fortnight, but in certain cases takes a longer course, with frequent relapses and short periods of remission. Infiltrations are usually resorbed in a matter of 8-10 days, leaving no changes in their wake.

Eosinophilic pneumonia must be differentiated from bacterial pneumonia and tuberculous infiltrations accompanied by eosinophilia. The diagnosis is verified by a search for eosinophils in the sputum and the x-ray. Signs in favour of eosinophilic pneumonia are its quick onset and cessation and the presence of other allergic manifestations—urticaria, eczema, skin lesions accompanied by pruritus and skin tests with an allergen.

Treatment. If the course is rapid no special therapy is required. In view of the allergic character of the disease steroid hormones (cortisone, prednisolone), dimedrol, calcium chloride, and anthelminthics must be administered instead of antibiotics.

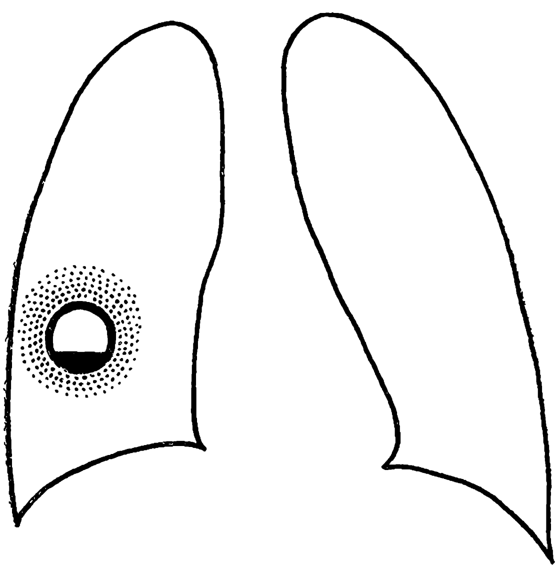


Fig. 30. Pulmonary abscess

LUNG ABSCESS (ABSCESSUS PULMONUM)

Lung abscess is characterized by a purulent liquefaction of the pulmonary parenchyma with a tendency toward delimitation from the normal pulmonary tissue. In the majority of cases, an abscess in its final stage forms a communication with a bronchus through which it then discharges its contents.

Aetiology and pathogenesis. Lung abscess (Fig. 30) is caused by pneumococci, streptococci, staphylococci and other organisms which gain entrance into the lung through the bronchi (bronchogenous infection) and with the blood stream (haematogenous infection). Lung abscess is usually secondary to acute pneumonia, particularly influenzal pneumonia, which severely debilitates the sufferer and causes lesions of the vessels, bronchi and alveoli. The second most frequent predisposing disease is aspiration pneumonia occurring in persons in an unconscious state or with deglutition disorders. It results from inspiration of mucus, blood, pus, vomit, pieces of tumours and tissue during operations on the oral cavity and the nasopharynx, and from aspiration of foreign bodies (tooth crowns, fruit stones, fragments of nut shells, etc.), which produce a collapse (atelectasis) in the pulmonary tissue and give rise to a purulent inflammation. In patients with haematogenous abscesses, pyogenic microbes are carried from septic foci with the blood stream into the lung, where they produce multiple abscesses of an embolic character. Lung abscess may be secondary to tuberculous pneumonia and suppuration of a pulmonary cyst, chest injuries, actinomycosis, hydatid cysts of the lung, pulmonary infarction, and bronchiectasis, since the presence of pus in the bronchi provides favourable conditions for the development of abscessed pneumonia.

Pathologic anatomy. In the stage of grey hepatization of the lungs attended by an intensified enzymatic proteolytic process, lysis affects not only the exudate but also the pulmonary tissue itself. In influenzal pneumonia, the alveoli division walls crumble down and the lung tissue becomes necrotized to be followed by abscess formation. Areas of necrosis in the lungs are also found after mustard gas and phosgene poisoning. Infection of the necrotized portions of lung tissue leads to the formation of abscesses and cavities surrounded by a dense fibrous tissue. The rupture of an abscess through a bronchus leaves a scar at the abscess site. In other cases, the cavity persists, and is periodically evacuated and filled with purulent matter. This is a chronic abscess which gives rise to new lung abscesses and amyloid degeneration.

Clinical picture. The onset of an abscess depends on the initiating factor. In cases where an abscess develops as a complication of pneumonia there is a gradual deterioration of the patient's general condition. Sometimes, an abscess begins with a shaking chill, pain on the affected side, fever rising to 39-40°C, and loss of appetite. The fever runs a remittent course. There is profuse perspiration at night, dry cough, and a considerable neutrophilic leucocytosis (up to 20,000 per cu mm blood) with a leftward shift and an elevated ESR. Both fever and leucocytosis depend on the presence of pus in the cavity. When the latter is evacuated, the temperature drops and the white cell count returns to normal. Their repeated increase is noted upon retention of pus in the cavity.

Percussion and auscultation findings depend on the depth of location and size of the abscess. Deep-seated and small abscesses are undetectable by percussion and auscultation. With an abscess of a considerable size, a dull percussion note is elicited from the area of infiltration, and a small number of medium and fine vesicular rales are heard there on auscultation. Amphoric breath sounds are heard over large, smooth-walled cavities.

Lung abscess is known to run its course in two periods: that before and that after its rupture into a bronchus. The length of the first period varies with different cases. The second period begins from the moment of abscess rupture into a bronchus with a copious sputum discharge (0.5 litre and more). When allowed to stand, the sputum divides into two layers: the upper—liquid and dark-green, and the lower—thick, creamy, green and odourless. Microscopy of the sputum reveals disintegrated cells, pus corpuscles, elastic fibres, drops of fat, needles of fatty acids, and numerous bacteria, the sputum sometimes being bloody and tenacious. After a rupture of sputum into a bronchus, the body temperature returns to normal, with amelioration in the patient's general condition. The sputum discharge is progressively reduced in quantity, the appetite is regained, the differential white cell count returns to normal, and the ESR is decelerated.

Sputum discharge depends on the position of the patient and abscess location. It is expectorated easier when the patient is lying on the unaffected side. With an abscess located in the upper lobes a vertical position is more helpful and for that in the lower lobes Quincke's position is best, i.e., when the foot of the bed is raised by 25 cm and the head end lowered. In the second period, i.e., after evacuation of the cavity, there is a delay in the respiratory movements on the affected side and the same physical symptoms as those found before abscess rupture into a bronchus. The rupture of an abscess into a bronchus may be followed by alternating periods of amelioration and aggravation of subjective symptoms. Most commonly, a deterioration results from occlusion of the draining bronchus and retention of the purulent contents of the abscess, with later absorption of its products by the blood.

In the acute period, slight albuminuria and haematuria appear. A person with a chronic abscess often has clubbed fingers and sometimes toes. The kidneys may undergo amyloid degeneration.

X-ray findings are highly valuable for the diagnosis of abscess. Before cavity formation there is only a diffuse shadow. The formation of the cavity and evacuation of its purulent contents, as well as resorption of pneumonic infiltration are shown by a distinct radiologic clearing. A nonevacuated abscess gives a spherical shadow. In the presence of liquid or air in the cavity there is a distinctly outlined shadow with a clear-cut horizontal level above the clearing.

Outcomes and complications of abscess. Most abscesses respond to therapy and resolve in complete recovery within 4-5 weeks of effective treatment. Lung abscess in a debilitated patient readily takes a chronic course. Among the complications of abscess, mention should be made of pulmonary haemorrhage, reactive serous pleurisy, and pyopneumothorax. Pulmonary haemorrhage due to an abscess is usually seen as an admixture of blood in sputum or as blood clots. Haemorrhages threatening life are comparatively rare in patients with an abscess. Complications on the part of the pleura consist of reactive ("sympathetic") serous pleurisy and pleural empyema, which is manifested by acute pain on the affected side, cough and remittent fever. In such cases, pleural puncture and examination of aspirated fluid are highly important.

Pyopneumothorax is a formidable complication of lung abscess. It results from rupture of an abscess into the pleural cavity, which is indicated by a sudden sharp pain in the side, accompanied by collapse, sometimes with a fatal outcome, resulting from the penetration of the purulent contents together with air to the extensive pleural surface with its numerous nerve endings. Pyopneumothorax is characterized by a recurrent abrupt rise of temperature and expectoration of a large amount of sputum of a creamy consistency. The percussion note on the affected side is usually dulled, and a delay in respiratory movements is markedly notable.

Differential diagnosis. Lung abscess should be differentiated from pulmonary gangrene. The latter produces a characteristically foetid sputum. A tuberculous cavern may give the same symptoms as a lung abscess; the presence in the sputum of tuberculous bacteria, however, as well as x-ray findings rule out the possibility of an abscess. Great difficulties are encountered in making a differential diagnosis between lung abscess and encysted empyema, especially its interlobar form.

Treatment and prophylaxis. The treatment of pulmonary suppurations is carried out by two methods: conservative and surgical.

A patient treated by the conservative method is at the onset of the disease strictly confined to bed, measures being taken to ensure a constant indraught of fresh air to the ward. The position of the body should facilitate free sputum discharge. The patient is given sulpha drugs (6-8 g daily), as well as antibiotics to aid resorption of the inflammatory infiltration even before the onset of the suppurative stage. From a purulent cavity communicating with a bronchus, the purulent contents are aspirated through a bronchoscope, and later a penicillin solution is injected directly into the bronchus. In addition, antibiotics are administered, in particular penicillin in doses of 250,000 U 2-3 times daily and streptomycin in doses of 0.5 g twice daily.

A remedy for a severe cough is codeine, as well as intravenous injections of a 10 per cent calcium chloride solution in a dose of 10 ml, and creosote, guaiacol and terpin hydrate (formulas Nos. 62, 63, 64) given per os. An amelioration is also achieved by a repeated blood transfusion in a dose of 100 ml. The patient's diet must be rich in proteins and vitamins, mainly vitamins A and C. During the initial 4-5 day period, 400-500 ml of liquid is given daily (dry diet).

The indications for surgical treatment of a lung abscess are as follows: (a) failure of the disease to respond to conservative treatment during a period of 4-5 weeks; (b) rupture of an abscess into the pleural space; (c) a severe course of lung abscess when high fever persists for one or two weeks, and the patient is emaciated.

Among the prophylactic measures the most important is a regular and timely control of influenza and pneumonia.

PULMONARY GANGRENE (GANGRAENA PULMONUM)

Pulmonary gangrene is characterized by a putrid destruction of the lung parenchyma and, as distinct from lung abscess, has no clear-cut anatomic boundaries.

Aetiology and pathogenesis. The causative organism of pulmonary gangrene is an anaerobic streptococcus. Histologic examination of a lung affected by gangrene reveals fusiform bacilli and spirochaetes, as well as *Streptothrix* and *Leptothrix* organisms; these, however, cannot be regarded as the specific agents responsible for gangrene. Very important are the character of the microflora, its virulence, capacity for entering tissues, for reproduction and existence in a given environment. The varied outcomes of an inflammatory process, however, rather than be determined by the composition of microflora, are mainly accounted for by the resistance of the body, its reactivity, i.e., capacity to resist the harmful influence of infection and defend itself. With a normal body resistance the normal tissue is readily delimited from the inflamed area by a demarcating line. If the resistance of the body is reduced, however, the inflammatory process grows worse, causing destruction of the lung parenchyma without enclosing the focus with a connective tissue membrane.

This leads to the emergence of pulmonary gangrene with a putrefactive microflora in sputum.

The causative organisms of gangrene gain entrance to the lungs by way of the bronchi and the blood stream (bronchogenous and haematogenous infections respectively), as well by direct extension from the adjacent organs undergoing putrefaction (contagious route).

Bronchogenous pulmonary gangrene is found in more than 90 per cent of all cases of gangrene. It develops as a result of the entry of anaerobic organisms inhabiting the oral cavity into the lungs by way of the respiratory passages. The predisposing factors of pulmonary gangrene are alcoholism, undernourishment, diabetes mellitus, injury to the lung tissue, especially in persons exposed to health hazards of dust (stove-makers, plasterers, etc.). Other predisposing factors are a history of pulmonary disease, in particular, pneumonia followed by pneumosclerosis and bronchiectasis. Pulmonary gangrene may be encountered with lung cancer, since in occlusion of the bronchial tube by a tumour the bronchus cannot be freed from the anaerobic microbes present within it, while the decomposed tumour matter offers them a favourable nutrient medium.

Pulmonary gangrene may be incited by lodgement of foreign bodies (a tooth fragment, food particle, fish bone, etc.) in the trachea, which is especially frequent in drunken persons, during an epileptic seizure, and in mentally deranged persons.

Gangrene may follow various septic diseases of the female genitalia, ulceration of the shank, thrombophlebitis of the haemorrhoidal veins, gangrenous appendicitis, and operations on infected organs of the abdominal cavity. In such cases, the causal agents of gangrene are carried to the lungs through the pulmonary artery with the blood stream (haematogenous infection).

Contagious gangrene occurs as a result of the ingrowth of gangrenous tissue into the lung during septic processes developing in the oesophagus due to cancer or scar tissue formation after a burn with concentrated acid and alkaline solutions.

Pathologic anatomy. A gangrenous lung is of a dirty-green colour, with a putrid odour due to tissue death. There are multiple or isolated foci of gangrene. The pleura corresponding to the affected area is inflamed and covered with a fibrin coat. In fresh cases, the area of gangrene adjoins healthy tissue.

Clinical picture. Men are more susceptible than women. The right lung is more frequently affected than the left, and the lower lobe more often than the upper. Gangrene may run an acute or a chronic course. The acute form of gangrene comes on with a sudden rise of temperature to 39°C and higher, and is accompanied by a sharp chest pain, sometimes with a blood-streaked sputum discharge, less often with copious blood spitting. The fever of an irregular type persists within a range of 38-40°C, goes down when the disease process abates and again climbs up when it is activated. A temperature fall coincides with sputum discharge from the gangrenous cavity. In retention of sputum, the temperature begins to rise again. In a few days' time, the sputum discharge is so profuse as to fill the mouth, if the gangrenous cavity has a wide communication with a bronchus. Sputum acquires an offensive odour, like that of dead tissue or a decayed tooth. Kept in a laboratory glass, the sputum divides into three layers. The lower one on the bottom is a crumbly deposit consisting of tissue particles and Dittrich's plugs—dense masses of a yellowish, sometimes grey colour, the size of a millet, fatty acid crystals (margarine needles) and drops of neutral fat. The medium layer of sputum is liquid and serous, and the upper one mucopurulent and frothy. The sputum is of a dirty-grey or dirty-brown colour, and ranges in amount from 200 to 300 ml per 24 hours.

The patient tries to assume a posture in which no sputum is discharged, as it is accompanied by an agonizing cough. If the gangrenous cavity is located in the lower parts of the lung the patient takes a half-sitting posture; in case it is in the upper lung he usually lies on the affected side.

There is a shortened percussion note over the affected area, while vocal fremitus is intensified. Sonorous moist and dry rales of varied calibre, crepitation and harsh amphoric breathing sounds are heard on auscultation. There is tachycardia and muffled cardiac sounds. The liver is enlarged and tender on finger pressure. The blood displays hypochromic anaemia, neutrophilic leucocytosis with a leftward shift, while the ESR is considerably elevated.

Pulmonary gangrene takes a chronic course in pneumosclerosis, bronchiectasis, and gives the same symptoms as the acute form, though less severe. Patients with this disease have clubbed fingers and sometimes toes.

X-ray shows the presence of a gangrenous process as shadows of a variable size, with a cavity inside without even borders and with a horizontal-level fluid collection above it.

Complications occur in the form of dry or wet pleurisy (purulent, gangrenous). Rupture of a gangrenous cavity into the pleural sac gives rise to pyopneumothorax, with a sudden pain in the side. rapid deterioration of the general condition, severe dyspnoea, and displacement of the heart toward the healthy side, with signs of collapse (cold perspiration, quick and feeble pulse, facial pallor, hypotonia, etc.). A profuse lethal haemorrhage takes place sometimes. Erosion of a large vessel by gangrene may lead to septicopyaemia. Other complications are general amyloidosis as well as amyloid degeneration of the kidneys, which is seen with the chronic form of gangrene.

Differential diagnosis is established between lung abscess, a tuberculous cavern, and bronchiectasis. Lung abscess produces no foetid sputum discharge. The sputum is purulent and is divided into two layers.

The x-ray picture, as distinct from that of gangrene, is one of a smooth-walled cavity with well-defined boundaries.

In case of a tuberculous cavern, the sputum is odourless and contains tubercule bacilli. The clinical picture is also characteristic.

It is more difficult to distinguish the clinical picture of gangrene from that of bronchiectasis. The diagnosis is aided by history-taking, the long duration of the disease process, and x-ray examination, especially bronchography. It is forbidden to undertake a diagnostic puncture of the gangrenous area, since this is likely to produce putrid pleurisy, phlegmon of the chest wall and sometimes a fatal "pleural shock".

Prognosis of acute gangrene has become comparatively brighter in view of the modern rational conservative or surgical treatment.

Treatment. The patient is strictly confined to bed. Intravenous injections of neosalvarsan (novarsenol, see formula No. 189) are given in a dose of 0.15-0.3 g every five days. The total dosage per treatment course is 2-3 g. Each infusion should be followed by urinalysis in view of the possible onset of haematuria, which signals the need for ending arsenic therapy. Blood-spitting is also a sign demanding an end to this treatment. Antibiotics (penicillin, biomycin, streptomycin, etc.) are used on a wide scale. If no amelioration takes place within 2-3 weeks surgery is required. Surgical intervention is doubtless indispensable for pyopneumothorax and purulent empyema. For anaemia, repeated blood transfusions in doses of 100-200 ml are undertaken. After a successful treatment, the patient will do wise to take a rest at a sanatorium or holiday home, bearing in mind the danger of a relapse. A change of climate has a beneficial effect on the patient's condition in later life. Preference should be given to regions with a warm climate, without drastic variations in the air temperature and atmospheric pressure. Prophylactic measures are taken to improve the general condition of the oral cavity.

BRONCHIAL AND LUNG CARCINOMA (CANCER PULMONUM)

Lung cancer may be primary and secondary, or metastatic, i.e., developing in the lung parenchyma by extension from other affected organs. According to statistical evidence for the past few years, cases of primary bronchial and pulmonary cancer have become more frequent and their share among other cancer diseases has noticeably increased. In the records of the incidence of malignant tumours of various organs, lung cancer in men ranked third and has now moved to the second place. The larger evidence on the occurrence of lung cancer on record over the past ten years is due not only to the actual increase in morbidity but also to the application of new diagnostic methods—bronchography, bronchoscopy, and tomography of the lungs. Lung cancer is the most frequent at the ages of 40 to 70 years, and is found in men 3-4 times more often than in women. Primary lung cancer usually affects the upper lobe and perihilar region, less often the lower lobe and rarely the middle lobe.

Aetiology. There is no reliable evidence on the causes of lung cancer. A number of observations, however, show the existence of a possible relationship between lung cancer and the presence in the inhaled air of various irritants—cobalt, nickel, arsenic, iron, radioactive uranium resin, motor engine exhaust, products of incomplete coal combustion, particles of asphalt tar, etc. All these substances contain cancerogenous elements. Findings of public polls show that lung cancer is much less frequent in non-smokers. Other factors contributing in some measure to the development of cancer are chronic inflammations of the lungs—nonspecific pneumonias, chronic bronchitis, bronchiectasis, pleurisy, pulmonary tuberculosis, chest injuries, particularly in wartime, and the presence of foreign bodies in the lung.

Pathologic anatomy. Carcinoma develops from the glandular epithelium of the primary and secondary bronchi, and is most commonly localized in the region of the hilus. Less frequently, carcinoma grows from the epithelial lining of the smaller bronchi and alveoli. The latter form of cancer runs its course over a lengthy period, without compressing the bronchi.

In respect of its microscopic structure, carcinoma occurs in the following three types: squamous cell carcinoma, microcellular epithelial carcinoma and adenocarcinoma. The most common is the nodular (mass) form of the tumour while the infiltrative form occurs less frequently. Within a large bronchial tube the tumour has the shape of a polyp and obstructs the lumen, disturbing bronchial patency. A tumour sometimes grows outwards and invades the adjoining tissues and organs, compressing the oesophagus, the trachea, and the recurrent and diaphragmatic nerves. Occlusion of a lobar bronchus causes collapse (atelectasis) of the entire lobe. At the same time, the lung is reduced in volume, while the mediastinal organs are displaced toward the site of affection, and the cupola of the diaphragm is drawn upwards. Disturbance of bronchial drainage causes multiplication of the bacterial flora at the site of the tumour and atelectasis, and is often accompanied by bronchitis, pneumonia, and suppurative processes in the lungs (abscess, gangrene, etc.). The growth of a tumour into blood vessels with their destruction entails bleeding. Lung cancer is prone to metastasize by way of both the lymphatic and blood vessels. Metastases occur in the regional lymphatic glands as well as in the supraclavicular, axillary, and cervical lymph nodes. They also develop in the pleura, attended by haemorrhagic pleuritis, in the other lung, the liver, the adrenals with the development of Addison's disease, bones, bone marrow, the spinal column, and ribs, with their destruction, which leads to spontaneous fractures. Metastases to the spinal column compress the nerve roots and cause severe pains, while metastases to the short and thick bones are accompanied by the appearance in the blood of juvenile cells of the myeloid group (leukaemoid reaction).

Microcellular carcinoma is particularly rapid in growth, early invades the adjoining organs and produces multiple metastases. Adenocarcinoma and especially squamous cell carcinoma run a comparatively benign course, insidiously and with late metastasization. Carcinoma localized in the lung periphery also grows slowly and for a long time gives no clinical manifestations. The infiltrative form of cancer often originates from the peripheral bronchi as well as from the alveolar epithelium and for its course resembles inflammatory infiltration. Decomposition of pulmonary tissue with inflammatory and suppurative processes is a fairly frequent occurrence. Carcinoma issuing from the alveolar epithelium or from the epithelium of the smaller bronchi is confined to the lung periphery and is round in shape.

Clinical picture. The initial and most frequent sign of lung cancer is coughing, which is consequent upon the growth of a tumour into the bronchial tube and accompanying bronchitis. This cough is often convulsive, like that seen in whooping cough, as a result of irritation of the region of the bronchial bifurcation. The cough is at first dry, and then is attended with a mucous and mucopurulent discharge, partly blood-streaked. Profuse haemorrhage is also seen sometimes, as well as daily blood-spitting, which very rarely looks like raspberry jelly. A frequent complaint is also pain felt in the affected side of the chest and growing worse in respiration. It irradiates to the shoulder and neck, and is excrusiating when emanating from the ribs and vertebrae affected by the tumour, because of the compression of the nerve roots. The patient complains of breathlessness, whose intensity depends on the size of a tumour and the degree of compression of the trachea and the principal bronchi. In this condition, respiration may become stenotic (stridorous). In compression of the laryngeal nerve the voice becomes husky. Fever is an inconstant though frequent symptom in lung cancer. It is produced by the accompanying inflammatory and suppurative processes occurring in the lungs and the pleura (pleurisy, pneumonia, abscess, gangrene, etc.), as well as absorption of the products of the necrotized lung tissue.

Inspection of the thorax may reveal certain signs characteristic of lung cancer. The affected side of the thorax is sunken and shows a delay in respiration, while the intercostal spaces are flattened. An unusually developed subcutaneous venous network is noted on the same side, which is due to difficult blood drainage. Compression of the vena cava superior and the innominate vein by the tumour gives rise to stasis and cyanosis in the region of the neck, the head, and the upper half of the trunk, with the face becoming puffy. In extensive metastases to the cervical and axillary lymph nodes, which are discovered by palpation, there is oedema of the neck and arm, and intense pain caused by disturbance in lymph drainage and compression of the cervical nerve plexus. The fingers are sometimes clubbed. This is usually seen in lung cancer accompanied by pneumonia and bronchiectasis.

A number of valuable signs of lung cancer may be discovered by means of percussion and auscultation of the lungs. With a tumour of a considerable size and the resultant bronchial occlusion with pulmonary atelectasis the percussion note is dulled, while the respiratory sounds and vocal fremitus are weakened on auscultation. As a rule, no moist rales or bronchial respiration are heard in lung cancer. They are encountered only in infiltrative forms of lung cancer, which for its clinical symptoms resembles pneumonia, differing from it in its stubborn and protracted course, the absence of high fever, recurrent blood-spitting, and resistance to therapy.

The blood shows moderate anaemia of a hypochromic character, neutrophilic leucocytosis with a rod neutrophil shift, and an increase in the red cell count. Metastases to bones produce a leukaemoid reaction with the appearance of juvenile cells of the myeloid group. Examination of sputum reveals polynuclear cells of a malignant newgrowth in fresh unstained as well as in stained preparations preliminarily fixed and enclosed in celloidin. Another sign of diagnostic importance is the presence in sputum of an increased number of red blood cells and elastic fibres due to decomposition of the lung parenchyma. Cancer cells may also be discovered in washings of the contents of the affected bronchus.

The most important for identifying lung cancer is x-ray examination (including tomography) which sometimes very early shows the shadow of a tumour or its secondary signs such as atelectasis and cancerous lymphangitis. Early identification of lung cancer is also possible with the aid of bronchography if the tumour is localized in the bronchi of the first and second order. In this case one can see the tumour itself or its secondary signs in the form of whitish scars on the mucous membrane of the bronchi, constriction and deformation of the bronchi and bulging of their mucous membrane. A more reliable information on the presence of a tumour is given by the very same bronchoscopy, when performing biopsy of the suspected site of a newgrowth. Valuable findings on the presence of a tumour in the bronchial tube are obtained by bronchography with a contrast substance, when the latter is retained by the tumour and fails to spread further along the bronchus. In peripheral lung cancer bronchography is less important. It is used when ordinary frontal and lateral roentgenograms as well as tomography fail to throw light on the suspected presence of carcinoma in the lung.

Treatment. Attempts at therapy of bronchial cancer with radium introduced through a bronchoscope, electrocoagulation of the tumour as well as radium needle punctures at thoracotomy have proved unsuccessful. More often, the entire affected lung is excised (pneumonectomy). The best long-term postoperative results have been obtained in the treatment of squamous cell carcinoma. Among other remedies x-ray therapy is used, i.e., transcutaneous radiation of a primary tumour and its metastases with x-rays and gamma-rays. This therapy abates cough and relieves dyspnoea, i.e., symptoms of bronchial compression. It is advisable to combine radiotherapy with blood transfusion, treatment with iron preparations, vitamins, antibiotics, and a high-caloric diet. Radiotherapy should be discontinued in case of pulmonary haemorrhage, pleurisy, a rise in temperature, and anaemia. Long-term results show that a temporary amelioration is often followed by an aggravation and a fatal outcome. Radiotherapy is contraindicated for a considerable emaciation, large and disintegrating tumours, metastases to the visceral organs, active pulmonary tuberculosis, a high-grade fever, and severe anaemia (haemoglobin level below 40 units).

Symptomatic treatment consists in the administration of narcotic drugs to allay pains, oxygen for dyspnoea, and cardiac drugs, as well as antibiotics and sulphonamides for suppurative and inflammatory processes in the lungs.

Prophylaxis. For the prevention of lung cancer, the air in residential districts and occupational surroundings should be kept clean, for which vigorous measures are taken to control air pollution by industrial smoke, motor engine exhaust, and road tar dust. For an early detection of lung cancer it is important to carry out a mass x-ray fluorography of practically healthy persons—men above 40 years of age in patricular.

Effective treatment of acute and chronic inflammatory processes in the lungs and control of smoking are also necessary.

PULMONARY OEDEMA (OEDEMA PULMONUM)

Pulmonary oedema is characterized by a protein-rich serous fluid passing from the lung capillaries into the alveoli and small and large bronchi and the development of stasis in the pulmonary circulation.

Aetiology and pathogenesis. Pulmonary oedema is secondary to acute cardiac insufficiency, heart defects, myocardial lesions, nephritis, pulmonary emphysema as well as poisoning with nitric oxide, alcohol, ether, morphine, and war gases (phosgene and diphosgene). The latter damage the capillary and alveolar walls and disturb the interchange of gases in the lungs; a favourable outcome is followed by pneumosclerosis.

Pathologic anatomy. On section the lungs are found to contain a foamy fluid; the alveolar walls are swollen; the capillaries and alveoli are dilated, the interstitial tissue and alveoli are filled with a protein-rich serous fluid. Oedematous fluid is also found in the bronchi, and sometimes obstructs the air passages to cause death from asphyxia.

Clinical picture. The clinical findings in pulmonary oedema are severe breathlessness due to the air passages being occluded with fluid which interferes with normal interchange of gases between the alveolar air and the blood in the capillaries. Breathing becomes loud and gurgling, and is heard from a distance. There is cold perspiration. The skin, especially of the face and extremities, is cyanotic, the conscience is dimmed. Coughing is attended with expectoration of a foamy pink sputum. The lungs are inflated, and many moist rales of a varied calibre are heard on auscultation. The pulse is quick and feeble.

In toxic oedema the first signs of intoxication are irritation in the throat, dry cough, sensation of pressure in the chest, quick and shallow respiration. It is known to run its course by two stages: blue anoxia and grey anoxia.

Treatment. Emergency aid to a patient with pulmonary oedema consists in immediate blood-letting (500-600 ml) and measures to intensify the heart's action with a 20 per cent camphor solution injected in doses of 2-3 ml once in every three hours, a 10 per cent caffeine solution in 1 ml doses, intravenous infusions of 1 ml of a 0.05 per cent strophanthin solution in 20 ml of a 40 per cent glucose solution, and inhalations of oxygen repeated every 10-15 minutes. Circulation of the blood is improved by means of cupping glasses, mustard plasters, hot baths for the hands and feet, and application of heat to the extremities. Morphine is forbidden for fear of paralysis of the respiratory centre. For patients with toxic oedema blood-letting is contraindicated. In the stage of grey anoxia artificial respiration is forbidden. It is advisable to give the patient an intravenous injection of a 10 per cent calcium chloride solution in a dose of 10 ml to reduce the permeability of capillaries.

PULMONARY INFARCTION (INFARCTUS PULMONUM)

Pulmonary infarction results from obstruction of branches of the pulmonary artery by a thrombus or an embolus brought from the right heart or from the veins of the general circulation. Pulmonary infarction is encountered in insufficiency of the right cardiac ventricle, inflammatory changes in the veins of the general circulation, thrombophlebitis of the lower extremities, puerperal infections, and septic abortions. In all such cases, clots of fibrin from the blood are deposited on the venous walls to form a thrombus which may be detached from the wall and carried with the blood stream to the right cardiac ventricle and further to the lungs, where it obstructs one of the branches of the pulmonary artery.

Much less frequently, pulmonary infarction develops from obstruction by a thrombus of the pulmonary artery itself. Obstruction of the main trunk of the pulmonary artery causes immediate death. Branches of the pulmonary artery may be obstructed also by air bubbles which entered the blood stream through injured veins, by globules of fat during bone fractures, and by camphor oil getting into a vein during injections.

In embolism of a branch of the pulmonary artery, the blood flow is stopped and changes develop in the wall of the obstructed artery. Plasma and erythrocytes penetrate from the thrombosed artery into the area supplied by it; the lung tissue is permeated with blood and takes a red colour, for which reason the infarction is called haemorrhagic. Small infarctions resolve without leaving a trace, whereas large ones become necrotized. The necrotic masses, whenever aseptic, are penetrated by scar tissue. In the case of an infected thrombus, infarction undergoes purulent (abscess) or putrid (gangrene) liquefaction. At x-ray examination, infarction is shaped as a pyramid, its base facing the pleura.

Clinical picture. Infarction is attended with a sudden acute pain on the affected side, a rise in temperature, breathlessness, blood-spitting, and a feeling of pressure in the chest. Infarction localized centrally and failing to reach the pleura gives no pain. The temperature does not exceed 39°C. On percussion, dulled sounds are heard over the site of infarction, while bronchial or vague breething sounds, small bubbling rales, crepitation and pleural friction sounds are heard on auscultation. Often an extensive infarction is accompanied by accumulation of inflammatory haemorrhagic fluid in the corresponding plural cavity. Local changes in the lungs disappear within 5-7 days of onset. In extensive infarction with pleural involvement the disease may take a protracted course. Recurrence of infarction, sometimes multiple, is possible.

Treatment. The patient is enjoined to have complete bed rest and take narcotic drugs to abate cough and pains: a 1 per cent solution of morphine or pantopon in a dose of 1 ml once or twice daily (formulas Nos. 103, 106 and 107), a 1 per cent dionine solution in a dose of eight drops, or codeine in powder form in a dose of 0.015 g two or three times daily.

It is necessary to take measures against diseases producing thrombi and embolism in thrombophlebitis, endocarditis, septic conditions, heart defects, etc. Prophylaxis is conducted in account with the pathogenesis of infarction. Particular caution should be displayed with regard to thrombus formation during vascular surgery. In the event of thrombophlebitis, before the thrombus becomes completely organized, one is recommended complete bed rest and anticoagulants (neodicoumarin, pelentan, phenylene—see formulas Nos. 29, 29a, 29b), keeping a check on the prothrombin time and carrying out urinalysis (haematuria).

PNEUMOCONIOSIS

Pneumoconioses are induced by prolonged inhalation of dust of a nonliving character. The most drastic changes in the lungs occur in conjunction with inhalation of mineral dusts of glass, enamels, cement, iron, lead, as well as coal dust. Pneumoconiosis resulting from exposure to coal dust is called anthracosis, that due to silica particles, silicosis, and that caused by iron, copper, and other metallic dust, siderosis.

In anthracosis the lung is black, in siderosis red, in exposure to tobacco dust yellow.

Dust particles lodged in the lymphatic glands, fissures, lungs, and endothelium of the lymphatic vessels produce irritation and proliferation of connective tissue with its later shrinkage and bronchial dilatation. The most common type of pneumoconiosis is silicosis, which usually occurs in conjunction with inhaling particles of silica contained in quartz or silicon oxide. Dust particles produce not only a mechanical but also a chemical damage, and are dissolved slowly, causing necrosis in the cell protoplasm. They are collected in the lymph spaces, around the bronchi and lymphatic vessels, and later cause peribronchitis, perivasculitis and scarring of the interstitial pulmonary tissue. Compression of vessels and bronchi brings about atelectasis, emphysema and bronchiectasis, especially in the lower lobes. Incising a lung affected by silicosis produces crackling sounds due to the presence of nodules, which may grow to the size of a pea.

Clinical picture. The patients's complaints in pneumoconiosis differ but little from those in pulmonary emphysema; when the disease is joined by cardiac insufficiency, there are complaints of a circulatory disorder. Physical examination reveals no serious abnormalities. The percussion note over the thorax may be clear, dulled or bandbox depending on the stage of the disease; on auscultation of the lungs, the breath sounds are diminished or somewhat harsh, with rales heard upon the supervention of bronchitis or tuberculosis. The x-ray shows an intense shadow of the lung roots, with patches and trabeculae resulting from the development of connective tissue.

In pneumoconiosis it is necessary to eliminate occupational hazards by introducing ventilation systems, humidifying the air, mechanizing manual jobs in mines, quarries, and dusty industrial surroundings. Another means to prevent pneumoconiosis is general advancement of the public sanitary standards, for example, provision of various public amenities in workers' settlements, strict observance of safety regulations in industry, etc.

PNEUMOSCLEROSIS (PULMONARY FIBROSIS)

Pneumosclerosis manifests itself in proliferation of connective tissue in the interalveolar and interlobular septa and in the bronchial walls, attended with respiratory dysfunction.

Aetiology and pathogenesis. Pneumosclerosis is secondary to acute and chronic inflammatory and noninflammatory processes in the air passages. This disease is fairly frequent after acute pneumonias—lobar, influenzal, measles, and whooping cough pneumonias, as well as after an abscess, pneumoconiosis, actinomycosis, and poisoning with gas and various chemical agents (irritants). Pneumosclerosis is also known to have followed wet pleurisy, tuberculosis of the bronchial lymph nodes, and traumatic lesions of the thorax attended by effusion of blood into the lung tissue. A particularly severe form of pneumosclerosis develops in connection with gunshot wounds of the lungs, complicated by haemo- and pyothorax. A frequent cause of pneumosclerosis is also chronic cardiac insufficiency. Inflammatory and noninflammatory processes, when extending to the lymphatic and blood vessels, the interstitial lung tissue and the peribronchial space cause a trophic (nutritional) disturbance in the lungs and bronchi, and give rise to pneumosclerosis. There occurs proliferation of the interstitial and connective tissue in the alveolar walls, between lobules, around the bronchi and vessels, and under the pleura. The respiratory, vascular (arteries, veins, capillaries) and lymphatic systems become involved in the disease process.

Pneumosclerosis disturbs pulmonary ventilation and the drainage (selfcleansing) of the bronchopulmonary apparatus. The disturbance in pulmonary ventilation causes deceleration of the blood and lymph flow in the affected area of the lung and intensifies the processes of sclerosis taking place there. Disturbance in the drainage of the bronchopulmonary apparatus entails bronchitis, which impairs the drainage (selfcleansing) function of the bronchi and tends to aggravate congestion and inflammation in the bronchi, with extension to the lung parenchyma. The collection of secretions obstructs the bronchus, with subsequent atelectasis, suppuration and scarring in the lungs. In affection of the alveolar walls the capillaries break down, which interferes with the pulmonary circulation and later causes hypertrophy of the right cardiac ventricle (cor pulmonale). In the early stages of pneumosclerosis, there is considerable dysfunction of the bronchopulmonary apparatus, in particular spasm of the branches of the bronchial tree, which results in an increased intra-alveolar and intrabronchial pressure. The latter entails loss of the bronchial and alveolar tone and the appearance of pulmonary emphysema and areas of bronchiectasis.

Clinical picture. The disease more frequently affects men above 40 years of age. In respect of its clinical course pneumosclerosis is subdivided into widespread (diffuse) and focal (segmentary). Diffuse pneumosclerosis runs its course as chronic bronchitis and bronchiolitis. The patient complains of a cough with a mucopurulent sputum expectorated with difficulty, later followed by attacks of dyspnoea. The face and the extremities are cyanotic, the face is puffy, and the thoracic cage is considerably inflated. Dry and moist rales are heard over the lungs, the left heart is enlarged, with a bulging arch of the pulmonary artery. There is periodic fever due to the development of lobular pneumonia and autointoxication. These pneumonias run a protracted course because of a disturbance in pulmonary ventilation and bronchial lymph drainage. Gradually, the disease process extends also to the pleura which grows thicker and undergoes deformation as a result of relapsing inflammations.

An invariable concurrent of pneumosclerosis is pulmonary emphysema, because the disease process extends to the smaller bronchi and bronchioles. This gives rise to cyanosis resulting from dysfunction of external respiration. Patients often die of lobular pneumonia, which runs a severe course because of a disturbed bronchopulmonary drainage and circulatory insufficiency. The amount of sputum is considerably increased. It is easily expectorated if pneumosclerosis is complicated by bronchiectases and sometimes acquires a foetid odour. The disease process culminates in cardiopulmonary insufficiency later followed by amyloidosis or amyloid-lipoid dystrophy of the kidneys.

The blood sometimes shows an increase in the amount of haemoglobin and the number of erythrocytes as a result of cardiopulmonary insufficiency and irritation of bone marrow. The white cell count and the ESR are normal, whereas an excess number of leucocytes and an elevated ESR point to an inflammatory process in the lungs. The disease runs a protracted course, and its outcome depends on the intervening complications and the condition of the heart.

Fulminant diffuse pneumosclerosis (Hamman and Rich disease). During the past few years, Hamman and Rich have reported cases of diffuse pneumosclerosis running an extremely rapid course. The disease ends fatally within a few months, with symptoms of cardiopulmonary insufficiency. Sometimes, the disease lasts 3-4 years and longer; therefore, some authors are inclined to regard such cases as subacute forms of diffuse pneumosclerosis.

The *aetiology* of this disease is unknown. A suggestion has been advanced concerning its viral or allergic origin. Evidence in favour of the latter is furnished by conditions found with the subacute form of pneumosclerosis, such as eosinophilia, scleroderma, rheumatoid polyarthritis, as well as by the efficacy of hormone therapy (corticosteroids, ACTH). In addition, morphologic changes in subacute diffuse pneumosclerosis are similar to those seen in periarteritis nodosa, lupus erythematosus disseminatus, rheumatism and rheumatoid polyarthritis.

Clinical picture. The characteristic symptoms are as follows: severe cyanosis and considerable dyspnoea caused by fibrous thickening of the alveolar walls which compress the capillaries. This causes a disturbance in oxygen diffusion and carbon dioxide release. The disease begins with an acute rise in temperature, dry cough or cough with a scanty mucous discharge, pains in the chest, and sometimes blood-spitting. Later, the fever abates, but dyspnoea, cyanosis, and debility become worse. Crepitation in the lungs is heard on auscultation. The symptom of clubbed fingers appears early. X-ray examination of the pulmonary interstices shows considerable infiltration, especially in the middle zone. Symptoms of cor pulmonale are often present. There is moderate leucocytosis, sometimes polycythaemia, and the ESR is accelerated.

The disease process may rapidly progress and end fatally; less often it takes a protracted course.

Treatment is symptomatic and is similar to that given for pulmonary abscess, bronchiectasis, emphysema, and cardiopulmonary insufficiency. In subacute pneumosclerosis, ACTH and corticosteroids are also used.

PLEURISY (PLEURITIS)

Aetiology and pathogenesis. Pleurisy, inflammation of the pleurae, is secondary to diseases of the organs coated by the pleura: the lungs, the mediastinum, and the lymphatic glands.

The most frequent is pleurisy of tuberculous, pneumonic and rheumatic aetiology. Pleurisy may also be traumatic in origin and may occur in diseases of the liver and the peritoneum, which communicate with the pleura through the lymphatic channels.

Tuberculous pleurisy is subdivided into two groups. The first group incorporates conditions which develop by extension of tuberculosis from the bronchi and the lymphatic glands directly or through the lymph and blood to the pleurae, with eruption of tubercles; the second includes allergic conditions resulting from tuberculous allergy. The latter group of pleurisy develop in exacerbations of a pre-existent tuberculosis of the lungs and lymph nodes as well as after a chill, acute infections, and fatigue, which play the role of a nonspecific irritant.

Pneumonic pleurisy also falls into two groups: parapneumonic and metapneumonic. The former occur simultaneously with pneumonia, the latter, which are sometimes purulent, come on after abatement of pneumonia. In addition, there is a small percentage of rheumatic pleurisy which is recognized from the presence of a rheumatic process in the heart, the joints, etc.

Pathologic anatomy. Pleurisy may be dry or wet. In dry pleurisy, the inflammatory exudate, rich in fibrin, coagulates on the pleura, so the latter is coated with sheets of fibrin. The pleurae lose their lustre and become thickened.

Wet pleurisy may be serous, serofibrinous, haemorrhagic, purulent, putrid and sometimes chylous and pseudochylous. Haemorrhagic exudate is encountered in pleurisy occurring with tuberculosis, cancer and scurvy, as well as with thoracic injuries and pulmonary infarction. Allergic and pneumonic pleurisy produces a serofibrinous exudation. Pleurisy may become purulent when microbes from a lung cavern or abscess gain entry to the pleural cavity; putrid—in pulmonary gangrene when pyogenic organisms enter the pleural cavity; chylous—in leukaemia, tumours, etc., as a result of rupture of the lacteal vessels; and pseudochylous—in persons with encapsulated pleural empyema. In wet pleurisy, the pleura is congested, the endothelium proliferates, the capillary walls become permeable, and the exudate accumulates in the pleural cavity. The pleurae are thickened owing to the implanted sheets of fibrin. In tuberculous affections of the pleura the latter is covered by tubercles which are subject to cheesy (caseous) decay.

When the exudate accumulates in the interlobar space and fails to effuse into the pleural cavity, interlobular pleurisy (interlobitis) comes on the stage. Prolonged pleurisy leads to the formation of commissures and pleural strands, the pleural surfaces sticking to one another, with possible areas of adhesion between the pleura and the diaphragm.

A collection in the pleural cavity of a noninflammatory fluid—transudate—is known as hydrothorax. Transudate differs from exudate in its specific gravity, protein content, and cellular composition. The specific gravity of exudate is 1,015-1,020, the protein content is equal to at least 3 per cent, the sediment contains leucocytes, erythrocytes, endothelial cells and cholesterol crystals. In transudate the percentage of protein does not exceed 2-2.5, the specific gravity is equal to 1 ,015, and no blood corpuscles are present in the sediment, which is extremely scanty. As distinct from exudate, transudate lies almost horizontally.

Dry Pleurisy (pleuritis sicca)

The onset of dry pleurisy is attended with sudden local pains in the chest, dry and agonizing cough, and fever. Pains grow worse on deep breathing, coughing, sneezing and motion, and compel the patient to lie on the affected side. Other distressing symptoms are debility, general malaise, anorexia (absence of appetite) and perspiration at night.

Objective examination reveals a delay in respiratory excursion, limitation of the mobility of the lung border on the involved side, sometimes a slightly dulled percussion note, which is due to a reflex tension of the muscles. A pleural friction sound is heard on auscultation, mostly in the lower parts of the thorax along the midaxillary line. The patient himself feels the pleural rub during a deep inspiration. The disease usually ends within a few days of the onset, and only in exceptional cases may it last as long as a fortnight. Dry pleurisy running a protracted course with periodic exacerbations is encountered with benign tuberculosis of the lungs or lymphatic glands.

Sometimes, dry pleurisy is mistaken for myositis, myalgia and intercostal neuralgia. The distinctive signs of the latter are absence of pains on deep inspiration and presence of points of tenderness on palpation.

Diaphragmatic dry pleurisy develops either with pneumonia in the inferior lobe or with diseases of the spleen or liver. The patient complains of pain in the region of the hypochondrium and the lower ribs, which becomes more intense on coughing, hiccough, eructation, and swallowing. The breathing may be shallow and costal, since the abdominal respiration is difficult due to irritation of the diaphragm. The mobility of the lower lung boundary and the diaphragm is limited. The breath sounds are unchanged. The x-ray shows the diaphragm in a high position and the sinuses insufficiently opened.

A patient with dry pleurisy should be strictly confined to bed until the abatement of fever, cough, and pain, whereupon a frugal life has to be led for another 7-10 days. Treatment is given mainly for the precursory disease responsible for dry pleurisy. Pains are allayed with codeine, heroine, cupping glasses, mustard plasters, strapping of the chest with a towel compress (warm dressing) or adhesive plaster for restricting movement. Aspirin is recommended in a dose of 0.5 g three times daily (formula No. 122).

Wet Pleurisy (pleuritis exudativa)

Wet pleurisy often arrives immediately in the wake of dry pleurisy and gives pain in the side, particularly on deep inspiration. There is a dry cough, general malaise, and a mild fever. Later, as fluid accumulates in the pleural cavity and the rough pleurae rubbing against each other become separated, the pain ceases, while fever climbs up to 39-40°C. In this period, the clinical picture of the disease is characterized by symptoms of a cardiovascular disturbance due to a high-grade fever and compression of the circulatory organs in case of considerable exudation. In certain cases, the disease is abrupt in onset, with a chill, a high-grade fever and rapid collection of exudate in the pleural cavity.

An important sign is a dry cough with a scanty mucous discharge. Cough is due either to accompanying bronchitis in the sunken parts of the lung, or is a manifestation of the principal disease in the lung. The patient's breathing is difficult, quick and shallow, with distressing breathlessness. Respiratory and circulatory disturbances occur mainly with considerable effusion resulting from a decrease in the respiratory surface, as well as compression and displacement of the adjacent organs. In right-side (dextral) effusion the heart is displaced to the left; in left-side (sinistral) effusion the heart is displaced to the right and its right boundary often follows the right midclavicular line. Left-side pleurisy causes depression of the diaphragm and the spleen; right-side pleurisy—of the liver. The veins on the neck are swollen as a result of compression of the vena cava superior, while compression of the vena cava inferior causes stasis in the liver; at the same time, the pulse is quickened, maybe to as high a rate as 120 beats per minute.

Fever which continues unabated with slight remissions in the morning sometimes becomes hectic, which points to the possibility of transition of serofibrinous pleurisy to pleural empyema. Notable on inspection are a bulging on the side with a considerable collection of exudate and a delay in respiratory excursion, while the intercostal spaces are flattened. Percussion of the thorax elicits sounds varying in quality with the amount of effusion and the site of percussion. At the site over the largest exudate collection the percussion note is dull, while in the other points it is muffled.

As distinct from hydrothorax, the inflammatory exudate in the pleural cavity follows an oblique rather than a straight line, and accumulates at first in the posterior and inferior regions, then moves upwards to the posterior axillary line from where it descends in the direction of the spinal column and forwards. The curvature obtained, which reflects the outline of the effusion is called Damoiseau's curve after the author who was the first to describe it. On the side of effusion, between Damoiseau's curve and the spinal column, there is a triangular area of tympanitic sounds known as Garland's triangle. With a large collection of exudate, percussion on the unaffected side may reveal a triangular area of dullness along the spinal column (Grocco-Rauchfuss' triangle) of which the apex adjoins that of Garland's triangle (Fig. 31). Dullness in Grocco-Rauchfuss' triangular space is due to pulmonary atelectasis on the normal side as well as resonance of the dull sounds from the side involved in the disease process. In left-side wet pleurisy Traube's space loses its common tympanitic note elicited on percussion to give place to a dull sound due to depression of the pleural cavity into this space (in case of copious pleural effusion).

The vocal fremitus and bronchophony are diminished. Over the effusion, where an area of atelectasis is usually found, one can hear breath sounds of a bronchial quality and intensified vocal fremitus, since the sounds from the larynx are better transmitted through a collapsed lung.

X-rays show a shadow the size and location of which depend on the extent of effusion. With a considerable effusion, the shadow follows an almost horizontal line. The x-ray also reveals a displacement of the mediastinal organs.

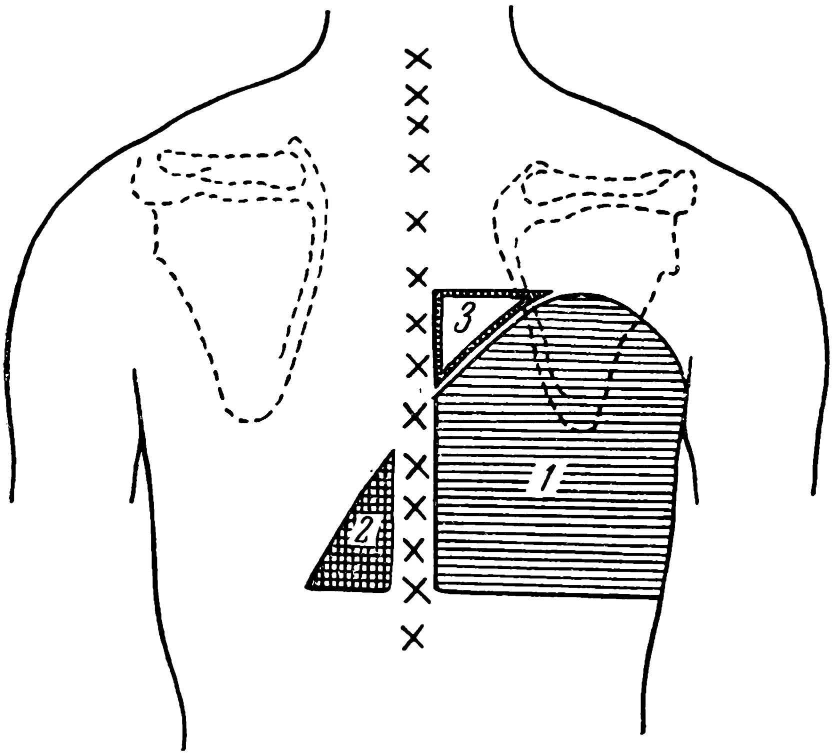


Fig. 31. Distribution of fluid in wet pleurisy

In all cases, the diagnosis necessarily rests on test puncture undertaken in order to establish the presence and ascertain the composition of fluid in the pleural cavity.

Pleural puncture is performed with a needle using a conventional 20 g syringe. The needle must be at least 7 cm in length and 1 mm in diameter. The site of puncture is in the point of maximal dullness or, which is preferable, in the eighth or ninth intercostal space along the posterior axillary or scapular line. Before undertaking a puncture its site and the surrounding field should be carefully disinfected at first with alcohol and then with tincture of iodine. The patient is seated on top of a chair, like in a saddle, preferably with the arm on the affected side raised and placed on the head or shoulder, because in this position the intercostal spaces are widened, which makes puncture easier. If the patient is hypersensitive and poorly tolerates pain the site of the puncture may be preliminarily anaesthesized with ethyl chloride (chlorethyl). The syringe is held in the hand like one would hold a fountain pen, and the needle is introduced along the upper edge of the underlying rib (Fig. 32). After aspirating a sufficient quantity of fluid, the needle is promptly withdrawn from the thorax, and the point of puncture is compressed with sterile cottonwool and dressed with a piece of gauze soaked in collodion. The fluid obtained is studied in a laboratory to determine its composition. Its colour, specific gravity, protein content, and cellular constituents are subject to determination.

In case of pneumonic effusion a smear from the effusion sediment shows prevalence of neutrophils over lymphocytes; in tuberculous effusion this relation is reversed. In purulent pleurisy, microscopy reveals leucocytes all over the sample and isolated erythrocytes.

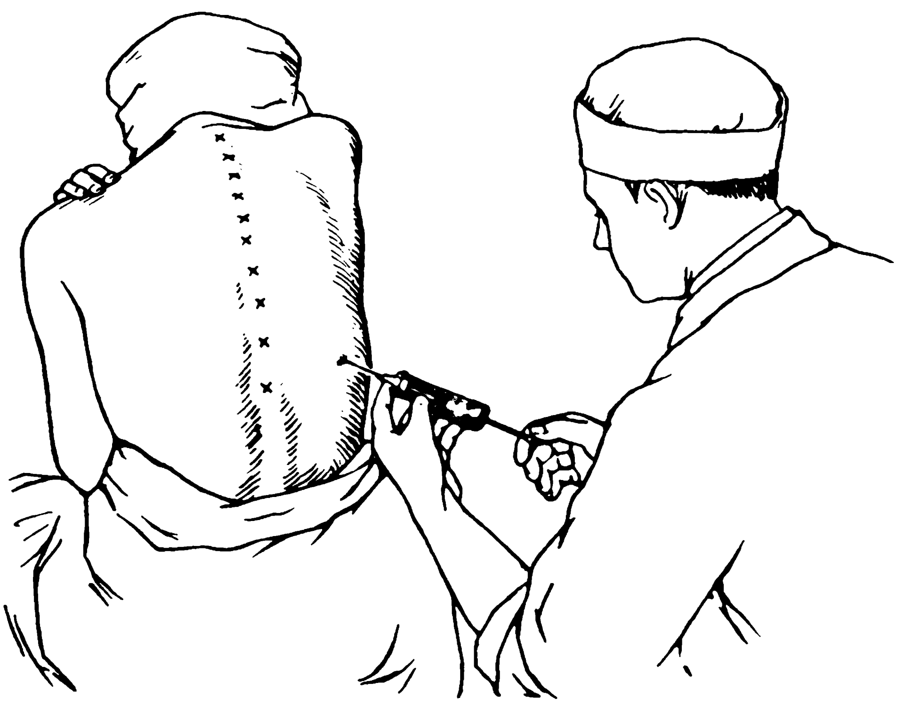


Fig. 32. Puncture of the pleural cavity

Mycobacterium tuberculosis is not discoverable in a sediment by the ordinary methods of smear staining; upon injection, however, of 10 ml of pleural fluid containing them into the abdominal cavity of a guinea pig, the latter dies of tuberculous peritonitis in 1-$1^1/_2$ month's time. In the presence of newgrowths, microscopy of the effusion shows endothelial and tumour cells.

The course and outcome of pleurisy. Tuberculous allergic pleurisy does not last longer than 4-6 weeks, and the fever usually begins to abate in one or two weeks' time. Rheumatic pleurisy passes within 5-10 days of onset; metapneumonic pleurisy continues for 10-15 days and resolves favourably if not complicated by suppuration. Lingering pleurisy always causes suspicions with regard to an active tuberculous process in the lungs. In pleurisy associated with cancer the prognosis is grave. In the majority of cases, pleurisy leaves connective tissue fibres. In case of considerable adhesions there is complete occlusion of the pleural cavities, while small adhesions cause limitation of the lung excursion at the sites of adhesion. In resorption of large collections of exudate the involved side of the thorax is sunken and obviously lags behind the normal side in respiration.

Treatment. The patient is strictly confined to bed. Pains are allayed with codeine, heroine, and applications of cupping glasses and mustard plasters. Sodium salicylate is administered per os in doses of 1 g three times daily or aspirin in doses of 0.5 g three times daily. Dry cough is abated with narcotic drugs. PASA should also be prescribed in doses of 2-3 g given four times daily before meals, with mineral or soda water.

In haematogenous disseminated forms of tuberculous pleurisy streptomycin is helpful in doses of 0.5-1 g daily. To promote urine excretion a 5 per cent calcium chloride solution is given in a dose of one tablespoonful daily or by an intravenous injection of a 10 per cent solution in a 10 ml dose. With a large effusion causing displacement of the heart and symptoms of cardiac insufficiency, it is necessary to aspirate the exudate. Aspiration is also undertaken in cases when the exudate fails to resorb for a long time after abatement of fever.

The aspiration is carried out with the aid of Potain's apparatus which is a graduated vessel of 1 litre capacity. The apparatus is provided with two rubber tubes. Into one of them is inserted the aspirating needle, while the other is connected to the vacuum pump. Taking all precautions listed above, the air is aspirated from the apparatus by means of the vacuum pump, and the tap at the end of the aspiration tube is closed. The next step is puncturing of the chest wall after which the tap is opened. During the aspirating procedure the needle is supported by the hand to avoid its displacement. It is recommended to draw not more than 1 litre of fluid at a time. To maintain cardiovascular function, the patient is given an injection of camphor prior to aspiration. If during aspiration the patient complains of dizziness and tinnitus, or if he grows pale and begins to cough violently, sometimes with a frothy sputum discharge, the aspiration should be stopped immediately, since these symptoms indicate irritation of the pleura and a severe disturbance in the blood circulation.

Highly important for the treatment of pleurisy is a sufficient diet with a rich vitamin content. In the period of convalescence, respiratory gymnastics is recommended to prevent adhesions. For stubborn pleurisy, failing to resolve for a long time, physiotherapeutic remedies are helpful, in particular quartz lamp irradiation and Sollux light in the case of residual pains. After recovery, a spell of sanatorial rest is advisable.

Pleural Empyema (Purulent Pleurisy, Pyothorax) (empyema pleurae)

The exudate becomes purulent as a result of the pleural cavity being invaded by streptococci, staphylococci, pneumococci and mixed infection from various suppurative foci in the body. A serous exudate sometimes turns purulent.

The disease runs a severe course with hectic fever rising to 39-40°C, the patient being wasted by chills followed by profuse perspiration. In objective examination the findings on percussion and auscultation of the chest are the same as those in wet pleurisy. The blood shows neutrophilic leucocytosis (20,000-30,000 per cu mm), a considerably elevated ESR, and anaemization which progresses in accordance with the gravity and duration of the disease. Spontaneous recovery occurs sometimes, when pus contained in the pleural cavity escapes by rupture into a bronchus, and is expectorated as a liquid creamy mass.

Pleural empyema bears very heavily on the condition of the cardiovascular system and the general tone of the body. The pulse is quickened, while languor and cachexia develop. In persistent cases, when pus is long retained in the pleural cavity and finds no way of escape, there is a danger of amyloid-lipoid degeneration of the kidneys with subsequent renal dysfunction.

Emergency aid in every case of pleural empyema consists of a puncture and evacuation of pus followed by injection into the pleural cavity of penicillin in a dose of 200,000-300,000 U. Simultaneously, penicillin is administered intramuscularly in a total daily dosage of at least 800,000 U, 100,000 U doses being given once in every three hours. Should penicillin therapy fail, surgery is necessary.

Pneumothorax

Pneumothorax is the presence of air in the pleural cavity. Pneumothorax occurs in pulmonary tuberculosis as a result of perforation of the pleura by a cavern, in suppurative processes in the lungs (abscess and gangrene), in thoracic injuries and pulmonary emphysema due to a rupture of the lung tissue on considerable physical exertion. Pneumothorax may be open, closed and valvular.

In an open pneumothorax, the opening formed in the pleura fails to close, so that during inspiration the air from the lungs enters the pleural cavity and during expiration escapes from the latter. An open pneumothorax is caused by thoracic injuries. In a closed pneumothorax there is no communication between the outer air and the pleural cavity.

Pneumothorax is called valvular when during inspiration the air from the lungs enters the pleural cavity, and during expiration fails to enter the lung from the pleural cavity, because the opening formed in the pleura is closed owing to an elevated intrapleural pressure. This results in a progressive accumulation of air in the pleural cavity. Pneumothorax is called artificial when it is induced for a therapeutic purpose in pulmonary tuberculosis.

In lung injuries blood enters the pleural space together with air during inspiration to give rise to haemopneumothorax. Seropneumothorax is a simultaneous collection in the pleural cavity of serous fluid and air, which is most commonly encountered in pulmonary tuberculosis. The presence in the pleural space of air and pus is known as pyopneumothorax, which results from a rupture of a lung abscess into the pleural space.

Clinical picture. Pneumothorax is characteristically acute or gradual in onset. In the former case, the patient complains of a sharp pain and breathlessness, and symptoms of collapse are present sometimes; the pulse is quickened and thready, and cold perspiration appears. The thoracic cage is inflated, the affected side notably bulges and lags behind in respiratory excursion, while the intercostal spaces are flattened. The vocal fremitus is diminished. There is a tympanitic percussion note on the affected side, while in a closed pneumothorax amphoric breathing is heard on auscultation.

With a simultaneous presence of air and horizontal-level fluid the percussion note in the lower parts is dulled, and a splashing sound is audible on shaking the chest.

The x-ray shows paleness in the lung shadow, displacement of the mediastinum, the heart, the stomach, and the liver, depending on the localization and extent of pneumothorax.

The course of pneumothorax depends on the underlying disease. A severe course is characteristic of (a) pneumothorax resulting from a rupture of a lung abscess into the pleural space; (b) valvular pneumothorax causing a grave respiratory or circulatory disturbance; (c) open pneumothorax complicated by pyopneumothorax.

Treatment. Bed rest is prescribed as well as morphine and oxygen inhalations to reduce breathlessness and pain. For a circulatory disorder the patient is given camphor (formula No. 31), and caffeine (formula No.32). In the event of a sharp displacement of the heart air is aspirated from the pleural cavity by means of pleural puncture. In the presence of haemo- and pyopneumothorax, aspiration of pus and blood from the pleural cavity should be followed by introduction of 200,000-300,000 U of penicillin into the pleural space. Open and valvular pneumothorax must be turned into a closed one by surgery.

PULMONARY TUBERCULOSIS

Pulmonary tuberculosis is caused by *Mycobacterium tuberculosis*, which produces characteristic tuberculous granulomas—tubercles—in the lungs.

This disease may also affect other organs: bones, joints, lymphatic glands, the kidneys, the peritoneum, etc.

The causative organism of tuberculosis was discovered by Koch in 1882.

Mycobacterium tuberculosis (Koch's bacilli) occurs in several types: (a) human (typus humanus), (b) bovine (typus bovinus), and (c) avian (typus avinus).

Human beings are infected mainly by mycobacteria of the first type.

The causal agent of tuberculosis is found, as mentioned above, in all organs and tissues of the sufferer, but its most favourite place is in the lungs and lymphatic glands. Tuberculosis organisms may also be found in sputum, urine, faeces, pleural exudate, and pus formed in joints affected by tuberculosis. Bovine-type bacilli most commonly infect persons employed on agricultural jobs who are likely to drink raw milk and eat insufficiently boiled meat of animals affected by tuberculosis. *Mycobacterium tuberculosis* contains a toxic substance known as endotoxin. The latter can be extracted from bacteria and passed through a Chamberland's filter. The extract is known as tuberculin. *Mycobacterium tuberculosis* is highly resistant, viable, particularly in dark and damp surroundings; however, it is very sensitive to the direct action of sunrays and high temperature. At a temperature of 80°C the organisms die within 5 minutes; a 5 per cent chloramine solution kills them within 6 hours, activated chloramine (a 2.5 per cent solution) in 2 hours, and chloride of lime within one hour. *Mycobacterium tuberculosis* is notable for its resistance to acids and alcohol.

The common source of tuberculosis infection is diseased person. The infection is airborne on droplets or spreads with dust as well as through the patient's personal belongings (handkerchiefs, bedding, blankets), upholstered furniture and various household utensils. The spread of infection is largely facilitated by contact with tuberculous persons or by living in a room formerly occupied by them. Infection of the gastrointestinal tract occurs mainly through milk from cows afflicted with tuberculosis.

Droplet infection takes place when a sick person's coughing, sneezing, and talking producesp rays and droplets containing *Mycobacterium tuberculosis*, which gain entry to the lungs of a healthy person with the air inhaled.

Dust infection occurs when sputum dries up and is taken up with dust. Dust particles containing tuberculosis bacilli may, during inspiration, enter the upper air passages and the lungs. True, the entry of mycobacteria into the system is not always followed by a disease. For an infection to become firmly implanted the following two prerequisites are needed: (a) sufficient activity and virulence of contagium; (b) susceptibility of the recipient to infection.

Pathologic Anatomy

The anatomic dislocation produced by tuberculosis is a tubercle (tuberculum) or a granuloma of a small size (up to 0.3 mm in diameter). A granuloma consists of epithelioid and giant cells surrounded by connective tissue cells. The latter form a dense capsule and separate the tuberculous focus from normal tissue. Tuberculosis organisms lodge within and between giant cells. The tubercle is subject to cheesy (caseous) degeneration. In the case of recovery, the tubercle gives place to scar tissue (fibrosis) or a deposition of lime salts occurs, i.e., it undergoes petrifaction (conversion into stonelike substance).

Development of the Disease

A tuberculous process is known to occur in two principal stages: (a) primary tuberculosis; and (b) secondary infection or reinfection tuberculosis. Primary tuberculosis is most commonly seen in childhood; it may, however, be encountered in juveniles and adults.

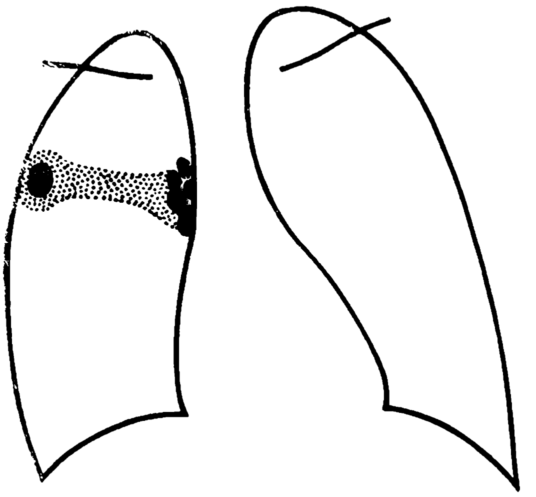


Fig. 33. Primary tuberculous process

In the overwhelming majority of cases, primary tuberculosis is aerogenous. It is a bronchopneumonic focus of tuberculosis lying under the pleura and surrounded by inflamed lung tissue (perifocal pneumonia). Such a focus is commonly called primary (Fig. 33). From the primary focus, tuberculosis organisms gain entry to the lymphatic vessels and regional lymph nodes in which they cause lymphangitis and a specific inflammation. The primary focus in the lung, together with lymphangitis and a regional affection of the lymph nodes, usually caseous in character, constitute the primary complex which undergoes changes during its development. In some cases, tuberculous pneumonia makes its appearance, in others tuberculosis organisms are carried to other tissues and organs with the lymph flow and blood stream and invade the entire system to produce what is known as miliary tuberculosis.

The primary complex most commonly tends to heal, i.e., the primary focus and corresponding lymph nodes become calcified, with the result and formation of the so-called Ghon's focus. After primary tuberculosis the sufferer develops hypersensitivity to the tuberculous toxin and acquires relative immunity (unsusceptibility) to tuberculosis infection.

Mycobacterium tuberculosis may survive for a long time. With unfavourable living conditions or a considerably reduced body resistance there is a risk of reinfection, i.e., recurrent infection with tuberculosis. In some cases, reinfection is due to the entrance of pathogenic organisms into the lungs through the airways (aerogenous or exogenous reinfection); in others, due to autoinfection, i.e., invasion by *Mycobacteria tuberculosis* retained in the primary complex. This source of reinfection is called endogenous, and in such cases the disease is precipitated by reactivation of long-standing tuberculous foci. The capsule of old calcified foci is loosened, with a fresh eruption of tubercles around them; the pulmonary tissue is softened and undergoes cheesy decomposition. When decomposition products get into a bronchus the disease process begins to extend bronchogenously. In other cases, decomposition products may escape into the lymphatic and blood vessels and contaminate all organs and tissues, i.e., produce dissemination of the tuberculous process.

Tuberculosis is known to run its course in two different forms: (a) exudative; (b) productive.

The exudative form is characterized by the appearance of a serous, catarrhal, or serofibrinous exudate in the alveoli. Pneumonic foci are prone to cheesy degeneration, decomposition, and formation of a cavity. The cheesy masses are partly resorbed and partly calcified.

The productive form of tuberculosis manifests itself by connective tissue proliferation, which entails shrinkage of the affected parts of the lung. In these forms, fibroblasts are also known to develop in addition to the epithelioid and giant cells, which contributes to a benign course of the disease.

Both these forms of tuberculosis may be present separately or in combination in one and the same patient, one of them being predominant.

Clinical Picture

In collecting evidence on diseases sustained in the past special attention must be paid to a history of dry and wet pleurisy, and relapsing and protracted febrile diseases which are often identified as influenza or a subfebrile condition of obscure aetiology. For a timely diagnosis of the initial forms of tuberculosis, a knowledge of the symptoms which sometimes mask the principal tuberculous process is necessary. For example, tachycardia and dyspeptic symptoms are very often the initial signs of tuberculosis. In certain cases, tuberculosis begins without giving any cause for complaints, and is diagnosed only by x-rays during prophylactic and group survey. This is partly due to the fact that in the early stages of the disease auscultation and percussion fail to reveal any morbid changes. At present, the common procedure in mass-scale prophylactic survey by x-rays is fluorography.

In the early stage of tuberculosis, the patient usually complains of general malaise, debility, early fatigue, irritability, partial loss of appetite, and emaciation, which are signs of intoxication. In the further course of the disease, there is cough, blood-spitting, and sweats at night, with general debility and emaciation.

Cough. Cough may be dry or productive, i.e., with sputum discharge, and depends for severity on the degree of anatomical damage to the lungs. The patient's excitability is also of certain importance. Coughing grows worse at night and in the morning, and is sometimes excruciating. In patients with cavities coughing is accompanied by a considerable sputum discharge. Coughing attended by hoarseness and pain on swallowing occurs in tuberculosis of the larynx.

Sputum. Sputum is mucopurulent. Microscopy shows a large number of pus corpuscles, erythrocytes, epithelial cells, elastic fibres and tuberculosis organisms. The latter's detection is a decisive factor in diagnosis, since the other elements may also be encountered with lung lesions of nontuberculous aetiology. In haematogenous forms of pulmonary tuberculosis, mycobacteria are often absent from the sputum.

Haemoptysis and Haemorrhage. Haemoptysis is sometimes the first sign of tuberculosis. In involvement of larger blood vessels it may become profuse to the degree of haemorrhage. Blood in the sputum is sometimes present as streaks. Cases of death immediately after a profuse haemorrhage are extremely rare. Haemorrhage may occur only once or repeatedly. It involves the danger of a further spread of infection if the infected blood finds its way into the unaffected parts of the lung.

Fever. This is one of the permanent symptoms of pulmonary tuberculosis, although it may be absent in the early period of the disease. Fever results from stimulation of the centre of thermal regulation with decomposition products of *Mycobacterium tuberculosis* and the affected tissues. Regular measurement of the patient's body temperature is of great practical importance for judgement on the character and course of the disease. In benign fibrotic processes, the body temperature is normal or subfebrile, even in the presence of cavities. In active forms, it may attain 38-39°C and sometimes becomes hectic, particularly in the presence of cheesy (caseous) degeneration in the lungs. A considerable rise in temperature is notable with pneumonic forms, when fever persists at a level of 38°C and higher for several months at a stretch. In haemoptysis and haemorrhage fever tends to aggravate. Perspiration at night in patients with pulmonary tuberculosis occurs fairly often, and is frequently an early sign of the disease. Cold profuse perspiration at night is sometimes evidence of a severe form of tuberculosis.

Dyspnoea. Dyspnoea is often seen in the final stages of pulmonary tuberculosis. It is due to cardiovascular insufficiency resulting from emphysema and fibrotic changes in the lungs. Dyspnoea is also concurrent with a widespread disease process in the lungs and with miliary pulmonary tuberculosis.

Emaciation. One of the typical signs of pulmonary tuberculosis is emaciation, for which reason this disease is also called consumption.

Emaciation is caused by tuberculous intoxication, a sharp increase in the metabolic rate and anorexia, and is notable mainly in progressive forms of the disease.

The gastrointestinal tract usually shows signs of dyspepsia, a decrease in gastric juice acidity, and flux (diarrhoea) which is consequent upon tuberculous intoxication.

Physical Methods of Examination

Inspection of the patient gives a number of valuable findings. The pallor of the skin, feverish blush on the cheeks, and shining eyes are all characteristic symptoms of this disease.

The asthenic body build is a frequent concomitant of and sequel to tuberculosis of the lungs, rather than its cause, as was erroneously considered by some authors, who regarded pulmonary tuberculosis as a disease of hereditary character. A sunken or narrowed chest is the result of pleural adhesions and cirrhotic changes in the lungs.

Palpation of symmetrical regions reveals tension and tenderness of the muscles of the shoulder girdle in the presence of fresh foci of consolidation (induration, infiltration), and in the case of a long-standing process, also flaccidness of the muscles. Hard, tender on palpation, and enlarged cervical, axillary, cubital and other lymph nodes are encountered with haematogenous forms of tuberculosis. Vocal fremitus is increased in consolidated lung tissue and reduced in wet pleurisy and pneumothorax.

Percussion may help detect changes in the lung parenchyma only where these changes are not deep-seated. A dull percussion note is characteristic of tuberculous pneumonic processes, severe fibrosis, and wet pleurisy, because in these conditions the amount of air contained in the alveoli is sharply reduced. In pulmonary emphysema and in the presence of cavities the percussion note is hollow or tympanitic in quality as a result of an increase in the amount of air in the lungs, lessening of tension and shrinkage of the pulmonary tissue. Percussion over large cavities often elicits tremulous sounds (cracked-pot sound). It is very important to determine the lower boundaries of the lung, because in pulmonary emphysema, effusion, and pleural adhesions, the excursion of the lung margins is markedly limited.

Auscultation of the lungs reveals changes in the respiratory murmur varying with the character of the tuberculous process. Coarse respiration is notable in fibrotic processes. Microfocal processes are characterized by a vague respiratory sound. Bronchial respiration is heard over the infiltrated areas. Weakened respiration is found with wet pleurisy, emphysema and extensive pleural adhesions.

Very important for diagnosis are rales, their character and sonority. One is advised to undertake auscultation after coughing at maximal inspiration, since in this case moist rales are heard more clearly. In the presence of cavities containing secretions, large bubbling moist rales are heard, and bronchial or amphoric breath sounds are audible over cavities communicating with the bronchi. Bronchophony may be intensified in consolidation of the lung tissue and is weakened in the presence of air or liquid in the pleural cavity.

In pneumonic forms of tuberculosis, there is moderate neutrophilic leucocytosis with a certain increase in the number of eosinophils and an elevated ESR. During fibrotic processes no neutrophilic shift is notable; there is lymphocytosis, eosinophilia, and a normal or slightly elevated ESR. The leukocyte count remains normal.

Diagnostic Importance of Cutaneous and Intracutaneous Tuberculin Tests (Pirquet's and Mantoux's Tests)

The tuberculin test is based on the reaction of the body to a subcutaneous and intracutaneous injection of tuberculin, which is an aqueous glycerol extract of a bouillon culture of *Mycobacterium tuberculosis.*

The reaction to tuberculin is ascertained by Pirquet's skin test and Mantoux's intracutaneous test, as well as by Koch's subcutaneous test.

The tuberculin test causes in infected individuals a rapidly disappearing hyperaemia (redness) at the site of tuberculin injection. With Mantoux's and Koch's tests, a general and focal reaction may develop in addition to the local one.

Pirquet's tuberculin test is of great diagnostic value only in early infancy, when a positive reaction is evidence of tuberculosis in the child. In adult individuals, a positive reaction is of no special significance, since most people have a record of tuberculous infection in childhood, so they may show a positive reaction even in the absence of an active tuberculous process.

For a Pirquet's test, a drop of pure tuberculin is applied to the skin on the inner side of the forearm, and a drop of a 25 per cent carbolic acid solution in physiologic saline is applied at a distance of 6 cm for control. The blunt end of a scalpel is used for scarification or incision of the skin, like that in smallpox vaccination, at first in the control drop and then in the tuberculin drop. The reaction is positive if a papule with a hyperaemic halo appears at the site of scarification. At the site of the control no reaction is shown.

Mantoux's test is more accurate. It is usually undertaken, when Pirquet's test is negative, by introducing a 0.1 ml dose of tuberculin diluted 1 : 10,000 into the skin of the forearm. With a negative reaction, tuberculin in a large concentration (1 : 1,000) must be introduced by the same method in two days' time. The control reaction is performed with a 25 per cent carbolic acid solution prepared on physiologic saline. If the reaction is positive, the site of puncture will become hyperaemic as early as in 8 hours' time, with a swelling and sometimes even vesicles. The reaction reaches its climax in 24-48 hours, which is then followed by a reversal.

Classification of Tuberculosis

Pulmonary tuberculosis may run a severe or a mild course, being localized in both lungs or in one lung, in one or several pulmonary lobes. In the course of the disease, productive or exudative processes may prevail, with or without a characteristic continuous or periodic discharge of *Mycobacterium tuberculosis.*

Classification of tuberculosis is highly important for its therapy and prevention, as well as for continuity in the treatment of a particular patient.

A. Pulmonary Tuberculosis

1. Primary complex
2. Tuberculosis of the bronchial lymphatic glands
3. Acute miliary tuberculosis

4. Subacute and chronic disseminated pulmonary tuberculosis
5. Lobular pulmonary tuberculosis
6. Infiltrative pulmonary tuberculosis: (a) infiltrates; (b) infiltrations
7. Cheesy (caseous) pneumonia
8. Chronic fibrocaseous pulmonary tuberculosis
9. Pulmonary cirrhosis
10. Pleurisy

Characteristics of the Course

1. Phases of development: (a) infiltration; (b) necrosis (decomposition, decay), dissemination; (c) resorption; (d) consolidation, calcification
2. Extension and localization in lung fields in each lung separately
3. Degree of compensation: compensated (A), subcompensated (B), decompensated (C)
4. Bacilli discharge: BK+; BK—; BK± (periodic bacilli discharge)

B. Extrapulmonary Tuberculosis

1. Tuberculosis in the bones and joints
2. Tuberculosis of the lymphatic glands
3. Tuberculosis of the skin
4. Tuberculosis of serous membranes (pericarditis, peritonitis, polyserositis)
5. Tuberculous meningitis
6. Tuberculosis of the larynx
7. Tuberculosis of the intestines
8. Tuberculosis of the urogenital organs
9. Tuberculosis of other organs

For ascertaining the extension of the tuberculous process each lung is conventionally subdivided into three fields: the upper, the middle, and the lower, which are correspondingly designated by the figures 1, 2, 3. The numerator shows the extension of the disease process in the right lung, and the denominator in the left.

The tuberculous process varies in severity. Sometimes, the patient is little troubled by its symptoms and retains his working capacity. This is a compensated process. In other cases, there is a subfebrile temperature, sweats at night, malaise, nervous disorders, general debility and reduced working capacity. This is a subcompensated form. Finally, in a decompensated form, the disease process takes a severe course, with a marked intoxication, fever, a considerable loss of flesh, the patient being ridden to bed. A compensated process is designated by the letter A; subcompensated, by the letter B; and decompensated, by the letter C. A discharge of *Mycobacterium tuber-*

culosis with sputum points to an open form which is designated BK+, whereas the absence of mycobacteria in the sputum indicates a closed form designated BK—. The designation BK± is used in case of an intermittent discharge of bacilli. In this way, graphic symbols can be effectively used to describe with a large measure of accuracy not only the localization and extension of the tuberculous process in the lungs but also the degree of bacilli discharge and the severity of the disease process. For example, the formula $C\,\frac{2+3}{1}\,BK+$ indicates that the tuberculous process is decompensated, i.e., it runs its course with grave clinical manifestations—an open form with the disease process being localized in the middle and lower field of the right lung and the upper field of the left lung. The presence of a cavity in a lung is designated by the letters CV.

The Clinical Forms of Pulmonary Tuberculosis

Primary pulmonary tuberculosis. Primary tuberculosis usually begins in childhood and affects above all the lungs and the bronchial lymphatic glands of the lung roots. Very seldom primary lesions are found in the gastrointestinal tract, still less frequently in the nose and larynx. The primary tuberculous lesion most commonly gives no clinical manifestations, and is detectable only by a positive Pirquet's and Mantoux's test and x-ray examination. In certain cases, a primary tuberculous lesion is attended with languor, debility, loss of appetite, brief spells of fever, retardation of growth, loss of weight, and dyspeptic symptoms. Physical examination sometimes reveals consolidated areas of pulmonary tissue of varying density, a shorter percussion note and a small number of dry and fine bubbling moist rales. A favourable course of primary tuberculosis leads to the formation of a Ghon's focus. In adults, the primary complex runs the same course as that in children. In unfavourable cases, primary pulmonary tuberculosis may end in an extensive perifocal inflammation giving rise to caseous pneumonia.

Tuberculosis of the bronchial lymphatic glands. In childhood, in the period of origination of the primary complex the infection also invades the bronchial lymphatic glands. The child is languid, with a reduced appetite and often a cough.

There is pallor of the skin, a loss of flesh, a retardation of growth, and a subfebrile temperature. The mediastinal and peripheral lymphatic glands are enlarged. Local symptoms may follow a considerable enlargement of lymphatic glands. There may be a sign of anisocoria (on compression of the sympathetic nerve), and dilation of skin veins (on compression of the venous trunks of the mediastinum).

Percussion and auscultation are of little help to diagnosis; the only substantial evidence in favour of a disease is a positive tuberculin test, x-ray findings and subfebrile temperature, as well as the above-described local symptoms. The disease runs an especially severe course in infants. The prognosis for older children is brighter. Staying outdoors, body hardening measures, sufficient diet rich in vitamins, accommodation at daytime sanatoria and schools situated in forest areas have a favourable effect on the sufferer and contribute to an early recovery.

Acute miliary tuberculosis. Acute miliary tuberculosis (from Latin *milium*—millet) is one of the most severe forms of haematogenous dissemination. It is caused by rupture of a primary or reinfection caseous focus into the blood stream. It runs its course with a high-grade fever and manifest toxaemia. There is an eruption of small tubercles along the course of the blood vessels in the lungs, in the brain meninges, on the ocular fundus and in all visceral organs and serous membranes. With regard to its clinical course, acute miliary tuberculosis is subdivided into typhoid, pulmonary and meningeal forms.

The typhoid form resembles enteric fever and is attended with a high-grade fever and cloudy consciousness. Widal's reaction in dilutions of 1 : 100 or 1 : 200 is positive. This form of tuberculosis, however, differs from typhoid in causing severe dyspnoea, cyanosis and tachycardia.

The pulmonary form of acute miliary tuberculosis runs its course with a high fever (up to 40°C), dyspnoea and cyanosis. Percussion elicits tympanitic sounds from the lungs; auscultation reveals weakened, harsh breath sounds and fine bubbling disseminated moist rales. The sputum contains no *Mycobacterium tuberculosis*. The x-ray shows uniformly arranged small focal shadows in the lungs.

The meningeal form is characteristically acute in onset, with a high temperature, severe headaches, slow pulse, rigid neck, positive Kernig's sign, cloudy consciousness, paralyses, and the presence of *Mycobacterium tuberculosis* in the cerebrospinal fluid. Acute miliary tuberculosis runs a violent course, and is accompanied by severe toxaemia. Timely streptomycin therapy may bring about recovery.

Chronic haematogenous-disseminated pulmonary tuberculosis develops in the same way as the acute form. The disease, however, takes a torpid and chronic course. It progresses insidiously or acutely and sometimes begins with wet and, more commonly, dry pleurisy and bronchitis. The periods of exacerbation alternate with remissions. During an exacerbation infiltrations are formed in the lungs which are subject to necrosis and may lead to cavity formation. The patient complains of a cough with sputum discharge which contains tuberculosis organisms. The ESR is elevated. The body temperature shows uncertian variations. Percussion elicits a normal sound with a tympanitic quality, while weakened and harsh breath sounds, dry sibilant rales, as well as sonorous moist rales and pleural rub sounds are heard on auscultation.

Chronic haematogenous-disseminated tuberculosis runs a benign course; usually, the infiltration resorbs with scar tissue formation (fibrosis). In certain cases, the disease is prone to progress, and effusion and lesions with multiple cavities develop in the lungs, the infection sometimes affecting the intestine, the kidneys, the larynx, etc. The x-ray shows gentle nonuniform and vague multiple focal shadows in the lungs.

Infiltrative pulmonary tuberculosis. The process of infiltration is provoked by external and internal causes. In the former case, the infection comes from without, during contact with bacilli carriers, in the latter case—from old foci, through the bronchi, the blood, and the lymph. The disease does not always begin in the same way. Sometimes, its onset coincides with a profuse pulmonary haemorrhage. Some of its forms are symptomless. In regard of its clinical course, infiltrative pulmonary tuberculosis most often resembles influenza and sometimes pneumonia. In all cases, however, persistent fever, a torpid course of the disease, a delay in resolution of pulmonary lesions (pneumonic forms), failure of sulpha drug and penicillin therapy should suggest a diagnosis of infiltrative tuberculosis.

Percussion of the lungs sometimes elicits a tympanitic sound and an insignificantly dulled percussion note, while auscultation reveals a weakened or harsh respiration with a bronchial note, dry rales, and sonorous medium bubbling moist rales in necrosis. Tuberculosis organisms and elastic fibres are discoverable in sputum only after tissue death. The body temperature ranges from 37.5 to 38° C. The ESR is elevated.

X-ray examination is exceedingly important for diagnosis, since auscultation and percussion reveal characteristic changes (dullness, bronchial respiration, moist rales) only in the event of extensive infiltration. In x-ray examination the lung tissue shows around homogenous shadow or diffuse cloudy shadows with clear areas in the centre in case of caseous degeneration.

Most commonly, infiltrative pulmonary tuberculosis has to be differentiated from nonspecific pneumonia which is more acute in onset and runs its course with an intensive neutrophilic leucocytosis. The onset of infiltrative tuberculosis is rarely acute. The infiltrate is usually confined to the upper lobes of the lungs, the disease runs a protracted course, and specific therapy is of little effect.

Lobular pulmonary tuberculosis. Lobular pulmonary tuberculosis is not a disease of definite pathogenesis. The foci may be soft or fibrous. Soft foci of tuberculosis are an initial manifestation after primary tuberculosis, fibrous foci result from a favourable outcome of other forms of pulmonary tuberculosis. Soft foci of tuberculosis are distinctive for their few symptoms. They must be detected by prophylactic surveys of the population. The clinical manifestations of fibrous foci of tuberculosis vary with the character and extension of anatomical changes in the lungs and the patient's reaction.

Soft foci of tuberculosis tend to progress in the majority of patients not given correct treatment in due time, whereas fibrous foci are much less prone to aggravate. The disease process is mainly confined to the apical, subclavian, parts of the lungs. Percussion over this area elicits a somewhat shorter percussion note and harsh breath sounds of a bronchial quality, and dry and moist rales of varied calibre. The x-ray shows at the lung apices microfocal shadows of variable density. Tuberculosis organisms are determinable in the sputum only after tissue death. The blood shows moderate leucocytosis with an insignificant leftward shift or lymphocytosis, while the ESR is elevated.

The disease process rarely becomes generalized. Its progression leads to infiltrative and then fibrocaseose tuberculosis.

Cheesy pneumonia. Cheesy or caseous pneumonia affects the entire lobe or separate lobules of the lungs. The disease has an acute onset (with chills, cough, and a rusty sputum discharge) and in this period resembles lobar pneumonia. Later, the temperature becomes hectic in character, and profuse perspiration sets in. The process is rapid in progress and extends to the larynx, the intestine, etc. X-ray examination reveals lobar shadowing of the lung with multiple paler sections. The sputum contains tubercle bacilli and elastic fibres. The blood picture is one of an elevated ESR, neutrophilic leucocytosis with a leftward shift, which is, however, less marked than in lobar pneumonia. On percussion there is dullness or tympanitic sound, while on auscultation there is a reduced respiratory sound with a bronchial quality and moist rales. The disease runs a very severe course and in the majority of cases its outlook is grim.

Chronic fibrocaseous pulmonary tuberculosis. Tuberculous necrosis produces pulmonary cavities, which are often encapsulated in a dense fibrous tissue, since, in addition to destructive processes, there is an intensified formation of fibrous tissue along the focus periphery. In such cases, the tuberculous process takes a benign course and lasts for years. In time, however, the disease grows worse, while continuous coughing, discharge of mycobacteria with sputum contribute to the spread of infection through the lungs, which in its turn causes periodic exacerbations of the disease process with a high-grade fever and sweats at night. These symptoms are joined by signs of chronic intoxication—tachycardia, dyspeptic symptoms, and impairment of appetite. The larynx and vocal cords often become involved also. The diagnosis rests on a history of past diseases, records of complaints, a shorter percussion note, the presence of bronchial or amphoric breath sounds, large bubbling sonorous rales, discovery of elastic fibres and mycobacteria in sputum, and x-ray findings which show large or small focal shadows as well as infiltration shadows with paler areas, distinctly outlined cavities and adhesive fibres.

Pulmonary cirrhosis is a considerable extension of a fibrotic process occurring in the lungs and is often accompanied by emphysema.

A circulatory disturbance is usually present, which is associated with cirrhosis (fibrosis) and emphysema.

The disease runs a chronic benign course, and is secondary to infiltrative and disseminated tuberculosis. The percussion note is dull and tympanitic. On auscultation there is harsh respiration with a bronchial quality and a considerable number of moist rales. The patient complains of breathlessness. There is a severe cyanosis, while blood-spitting and haemorrhages are fairly frequent; mycobacteria in the sputum are discovered rarely. The blood cell count and the ESR are unchanged. Death, as in heart disease, is often due to right ventricular insufficiency attended by symptoms of stasis in the liver, ascitis, etc.

Prophylaxis

Tuberculosis is world-wide in distribution. In 9 out of every 10 cases it affects the lung. Pulmonary tuberculosis occurs at any age, but it is more common in adults. Undernourishment, inadequate daylight and fresh air deficit, crammed, damp and unsanitary housing conditions are all factors contributing to the prevalence of tuberculosis.

In the Soviet Union, the rate of tuberculosis has drastically declined and continues to fall as a result of measures to promote public welfare, enforcement of labour safety legislation, and also special efforts to improve public health (provision of holiday homes, sanatoria, centres for tuberculosis control). An important role in reduction of morbidity is played by health education. A great contribution to the fight against tuberculosis is made by special centres for tuberculosis control known as dispensaries which are making efforts to wipe out this disease in the USSR. These centres carry out early detection, medical treatment and prevention of tuberculosis.

For an effective solution of its tasks, the centre for tuberculosis control carries out the following measures: (a) puts on record fresh cases and carries out treatment of all patients with tuberculosis, including those with primary symptoms, sends patients to hospitals for therapy; (b) carries out examination of the families of tuberculotics, watches over the health status of family members and the patient's living conditions, specally employing public health nurses for this purpose; (c) carries out mass-scale x-ray examination of various population groups—schoolchildren, personnel of child welfare centres and medical institutions, workers of the food industry and industries with occupational hazards; (d) conducts health education at the tuberculosis control centre, at home and at work by employment of public health nurses; (e) selects patients to be sent for treatment to sanatoria and health resorts and to night sanatoria; (f) keeps records of tuberculosis morbidity and death rate; (g) handles matters involved in rehabilitation of tuberculous persons and training them for a new profession; (h) transfers patients to part-time jobs.

In accordance with Soviet legislation, industrial managers and trade-union locals are obliged to employ tuberculous persons on easier jobs whenever the latter require it.

Another measure of public prophylaxis is vaccination. The French scientist Calmette and his associate Guérin cultivated bovine-type tubercle bacillus (*Mycobacterium*) on a medium with an admixture of bile and carried out multiple reinoculations on various media, as a result of which the bacilli lost their virulence and when inoculated to animals failed to cause any specific tuberculous lesions. These animals proved resistant to infection when repeatedly infected with *Mycobacterium tuberculosis.*

In order to increase the resistance of the child body to tuberculosis infection all newborn babies in the Soviet Union are given an injection of BCG vaccine suggested by Calmette and Guérin.

The application of this vaccine has shown that the immunity it confers lasts from 18 months to 2 years, after which time vaccination has to be repeated. The morbidity and mortality rates of tuberculosis among vaccinated persons are considerably lower than in unvaccinated population.

One of the principal means of checking the spread of tuberculosis is destruction of the causal organisms. For this purpose, sputum is accurately collected in special lid-covered cuspidors. Before they are given to patients, these spit receptacles are filled to one-third of capacity with a 5 per cent chloramine solution. After a cuspidor is fully filled with sputum, it is disinfected together with sputum with a 5 per cent chloramine solution allowed to remain in the cuspidor for six hours. Sputum may also be disinfected with dry chloride of lime at the rate of 2 g to 10 ml. The cuspidor should be filled with sputum to half its capacity. The easiest means of disinfecting a cuspidor is by boiling it in a 2 per cent soda solution. The patient's clothes should be immersed in the same solutions or in a corrosive sublimate solution. It is absolutely necessary to immerse the patient's towels and handkerchiefs in a disinfectant solution. Sputum must be disposed of into the sewerage system only after it has been disinfected. It is also indispensable to adhere to rules of personal hygiene at meals, have regular sleep and rest, systematically strengthen one's physical condition by hardening the body and light gymnastics. A baby borne of a mother with tuberculosis should immediately after birth be isolated from her and fed by artificial means.

Treatment

Tuberculosis responds to treatment started in good time and when the sufferer observes the required rules of hygiene and diet. Adequate nourishment is particularly important in this respect.

The patient's diet should include sufficient quantities of butter, milk, milk cream, food rich in proteins and carbohydrates, fruit, especially grapes, which contain much sugar, and vitamins.

The daily caloric intake must be equal to at least 3,000 Calories.

Tuberculous persons are advised to stay in the fresh air, take regular walks and sleep in the open air. Staying outdoors improves the patient's general condition, whets up the appetite and substantially fortifies the body defences against infection.

Kumiss treatment. Tuberculosis is effectively treated with kumiss. It is applicable, however, only to compensated and subcompensated forms of the disease, and is contraindicated for decompensated processes. Kumiss is fermented mare's or camel's milk which contains proteins, carbohydrates, lactic acid, milk sugar, alcohol (1-3 per cent), carbon dioxide, etc. Kumiss therapy is very efficacious in a steppe climate, particularly for pulmonary tuberculosis coexisting with chronic bronchitis and bronchiectasis. Kumiss is recommended in daily doses of 1-2 litres, two-thirds of the daily dose being taken in the morning and the remainder in the evening (between 4 and 7 p.m.).

Climatic, sanatorial and health resort treatment. Sanatorial and health resort therapy is the most helpful for compensated and subcompensated forms of tuberculosis. It is absolutely forbidden to send to resort areas patients with severe forms of pulmonary tuberculosis, with tuberculous lesions of the intestine, the larynx, and other organs. The favourable effect of sanatorial therapy on a tuberculous person is due to the rational hygienic and dietary regimen existing in sanatoria, prolonged exposure to fresh air, and highly nourishing food. In addition, the nervous system is strengthened under the influence of a change in environment, complete rest, and the beautiful scenery. At a sanatorium, patients take sun and air baths, which produce a beneficial effect on the circulation of the blood and cutaneous respiration. Solar therapy is forbidden in the presence of fever, marked general debility, and blood-spitting. At a sanatorium, the patients engage in light gymnastics and useful work.

Sanatorial treatment is, as a rule, recommended in the region of the patient's permanent residence.

Drug therapy. Dionine (ethyl morphine hydrochloride) and codeine are given for cough and chest pains. In the case of blood-spitting, the patient is enjoined to have complete rest and refrain from sharp movements, much talk and excitement. Food and liquid are taken cold and in a limited quantity. A helpful remedy against haemorrhage is a tablespoonful of common salt given with a half-glassful of water in order to promote blood coagulation. Another means to arrest haemorrhage is an intravenous infusion of a 10 per cent calcium chloride solution in a 10 ml dose or a 20 per cent common salt solution in the same dose. Administrations of morphine or pantopon are forbidden since after abatement of cough the blood invaded by mycobacteria may gain entrance to the normal parts of the lung and cause an extension of the disease process. In order to enhance blood coagulation, vitamin K (vicasol) is given in powder form three times daily (formulas Nos. 159 and 159*a*). For symptoms of cardiac failure, digitalis is given per os and camphor and caffeine by subcutaneous injections (formulas Nos. 1, 31, 32).

In case of sweats at night, the patient's room must be frequently ventilated. After a bout of perspiration the skin should be wiped dry and the clothes changed. Very helpful are daily sponging of the body with lukewarm water with an addition of vinegar or alcohol and a subcutaneous injection of 1 ml of a 0.1 per cent atropine solution. As a roborant remedy a subcutaneous injection of arsenic with strychnine as well as iron preparations are recommended. Bitters are given to improve the appetite (formulas Nos. 73 and 74). For a high-grade fever with symptoms of intoxication (headache, insomnia), very effective is peroral administration of small doses of pyramidon in combination with caffeine (formula No. 119).

During the past few years, considerable progress has been made in the treatment of pulmonary tuberculosis with antibiotics, streptomycin in particular (formula No.151). A course of streptomycin treatment lasts from 2 to 4 months. The patient is given daily intramuscular injections of streptomycin in doses of 0.5 to 1 g (1 g of streptomycin is equal to 1,000,000 U) in a physiologic salt solution. Streptomycin is sometimes liable to cause dermatitis, a visual disorder, dizziness, headache, and palpitation, which may necessitate its withdrawal.

Very efficacious for all forms of pulmonary tuberculosis is phthivazide (formula No. 193). Its 24-hour dose during the first ten days is 0.5 g, in the later period 1 g. Phthivazide is taken per os twice daily in equal doses. The course of treatment lasts from 2 to 4 months.

Phthivazide is contraindicated for stenocardia and dermatitis, because it is likely to provoke their exacerbation.

Tuberculosis also responds to therapy with PASA (paraaminosalicylic acid—formula No. 192). The patient is given from 10 to 12 g of the drug in 24 hours, the total 24-hour dose being divided into four equal portions. Treatment lasts from 2 to 4 months in combination with administrations of streptomycin or phthivazide. PASA is taken half-an-hour after meals with milk or alkaline water.

Surgery in pulmonary tuberculosis. Surgical treatment of pulmonary tuberculosis is used on a considerable scale.

Artificial pneumothorax. Pneumothorax essentially consists in artificial compression of the lung by injecting air into the pleural cavity (collapse therapy). After pneumothorax is induced in the presence of adhesions between the lungs and the parietal pleura, which interfere with retraction of the lung, the adhesions are broken down by cauterization. Indications for artificial pneumothorax are as follows: a fresh cavity, pulmonary haemorrhage, and lung tissue infiltration with invasion by tubercle bacteria. Pneumothorax is ineffective for extensive pleural adhesions, long-standing fibrotic processes, and a severe general condition of the patient. Injection of air into the pleural cavity is periodically repeated during several years so as to maintain sufficient pressure in the pleural cavity until a lasting effect is obtained. Artificial pneumothorax helps restore working capacity in 70-80 per cent of cases, and is one of the most effective remedies against tuberculosis.

Extrapleural pneumothorax. Another method of treatment is extrapleural pneumothorax, i.e., injection of air into a cavity formed by surgery between the endothoracic fascia and the parietal pleura. At operation, subperiosteal resection of one rib is performed above the affected lung lobe. This method is advantageous in that the lung is compressed only in the region of extrapleural pneumothorax, whereas the other parts of the lung remain open.

Thoracoplasty. For long-standing chronic cavitary processes attended with a large number of pleural adhesions, thoracoplasty is used. This operation consists in resection of a certain number of ribs, varying with the specific features and extension of the disease process, so as to produce retraction of the thoracic wall and compression of the lung with atmospheric air from the outside.

Fixation and a certain compression of the lung can be attained also by artificially induced paralysis of the diaphragm through alcoholization of the diaphragmatic nerve. This operation, however, is rarely used today. For cavities seated under the pleura, cavernotomy (opening of a cavity) is performed, followed by treatment of exposed tissue. When the patient is in a satisfactory general condition and the process of cavity formation is circumscribed, a segment or a lobe of the lung is resected. With an extensive process in one lung, the other lung being normal, total resection of the affected lung may be undertaken.

Tuberculosis and Pregnancy

In progressive forms of tuberculosis running their course with fever, blood-spitting, cavity formation, tubercle bacilli present in sputum, and general emaciation of the patient, pregnancy is contraindicated and should, therefore, be interrupted by artificial abortion.

Compensated forms of tuberculosis are not a contraindication for pregnancy.

Diseases of the Cardiovascular System

AN OUTLINE OF ANATOMY AND PHYSIOLOGY

The heart is located in the anterior mediastinum and is held in place by large blood vessels. Its greater part is to the left of the median line, the lesser part to the right, and it ranges in weight from 250 to 350 g. The heart is divided by a longitudinal septum into right and left halves, which in their turn are subdivided into the atria and ventricles. The atria communicate with the ventricles through the atrioventricular orifices. The heart has three sacs: the internal (endocardium), the middle (myocardium), and the external (pericardium).

The endocardium lines with endothelial cells the inner surface of the ventricular and atrial chambers. At the inlet and outlet openings of the cardiac cavities the endocardium is thickened, forming folds known as the heart valves. The valve between the left atrium and the left ventricle consists of two leaflets (cusps), and is called the bicuspid or mitral valve; the valve between the right atrium and the right ventricle is known as the tricuspid valve for the number of its leaflets. The blood from the left ventricle flows through an orifice into the aorta; a backflow of blood from the aorta into the left ventricle is prevented by the semilunar aortic valves. From the right ventricle the blood flows into the pulmonary artery, whose orifice is guarded by three semilunar valves, which also prevent a backflow of blood into the ventricle.

The middle muscular sac of the heart, called the myocardium, is unevenly developed in different parts of the heart; in the right ventricle it is less developed than in the left. The muscular layer of the ventricles is considerably thicker than that of the atria. The fibres of the heart muscle are intertwined. This facilitates an instantaneous excitation and contraction of the entire heart muscle. On its surface facing the cardiac cavity the muscular layer forms trabeculae as well as the papillary muscles connected to the margins of the tricuspid and mitral valves by fibrous bands, the chordae tendineae.

The external (serous) sac, or the pericardium, consists of two coats—the inner and the outer. The inner, or visceral, coat folds at the base of the heart to form another coat—the outer, or parietal. Between them, there is a cavity which constantly contains about 25 g of serous fluid.

The heart is the pumping apparatus of the circulatory system. The venae cava inferior and superior empty into the right atrium. The vena cava inferior collects venous blood from the organs of the abdominal cavity and the lower extremities, and the vena cava superior from the head, the neck, the organs of the thoracic cavity, and the upper extremities.

During a systole all venous blood contained in the right ventricle is ejected into the pulmonary artery, which ramifies and transforms into the capillary network lining the alveolar walls. Here is the site of gas interchange with the alveolar air. The vessels containing oxygenated blood finally form four pulmonary veins which empty into the left atrium. During a heart contraction, the blood is delivered from the left ventricle into the aorta. The section of the aorta above the diaphragm is known as thoracic aorta, that below the diaphragm, as the abdominal aorta. From the thoracic aorta, arteries branch off to the head and upper extremities (the carotid, the brachiocephalic trunk, and the left subclavian artery), and from the abdominal aorta to the abdominal organs and the lower extremities. The arteries ramifying into vessels of a smaller calibre end in capillaries of 7-8 μ in diameter. The capillaries consist of one layer of semipermeable endothelial cells. The blood flowing through the capillaries supplies tissues with oxygen and nutritive material—salts, water, sugar, amino acids, etc., and takes up waste matter subject to elimination from the body. The capillaries have two limbs, the arterial and the venous, which directly communicate with one another. Both limbs form a loop. Most of the capillaries (75 per cent of the total) are usually in a collapsed state, but during physical exertion they are filled with blood. The circulatory system is divided into the systemic (general) and the pulmonary (lesser) circulation. The part of the vascular system, from the exit of the pulmonary artery from the right ventricle to the entrance of the four pulmonary veins into the left atrium is known as the pulmonary circulation. The remaining vascular system, from the aorta and its ramifications to the venae cava superior and inferior, is called the systemic circulation. The blood flow through this system is constant and regular. The blood flow through the circulatory system is effected by the action of a number of factors, above all the uniform and periodic contraction of the heart. Excitation (impulses) causing heart contractions arises in the right atrium, near the mouth of the vena cava superior, in the Keith-Flack node (sinoatrial node). From this node impulses are conveyed to the Aschoff-Tawara node (atrioventricular node) in the atrial septum. From the Aschoff-Tawara node, impulses are transmitted

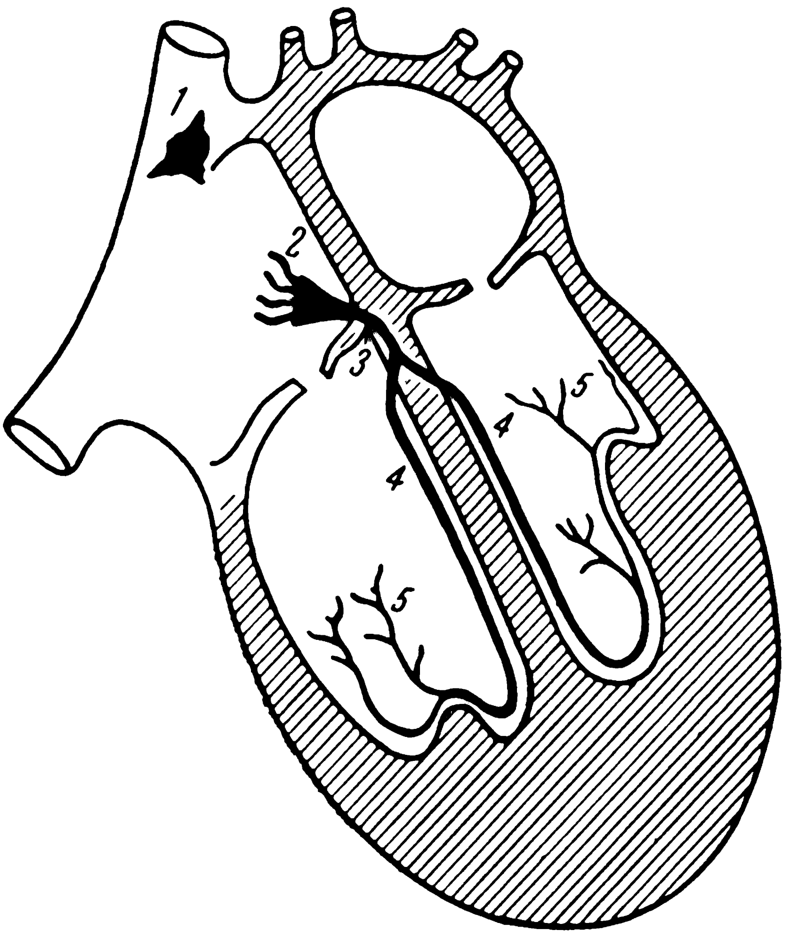


Fig. 34. Conducting system of the heart

1—Keith-Flack node; *2*—Aschoff-Tawara node; *3*—bundle of His; *4*—right and left branches of the bundle of His; *5*—Purkinje's fibres

to the bundle of His, which, after passing through the fibrous ring of the heart, ramifies into two branches. One of them descends into the right, the other into the left ventricle along the respective side of the ventricular septum. Both branches of the bundle of His terminate with Purkinje's fibres. The latter convey the impulse to the heart muscle, causing it to contract. The entire system through which impulses are conveyed, beginning with the Keith-Flack node and ending with Purkinje's fibres, is known as the conducting system, which consists of embryonal muscle fibers (Fig. 34).

Heart contractions show a certain regularity, occurring at an average rate of 70-80 beats per minute. Contractions at first occur in the atria and then in the ventricles.

On the average, the entire cycle of the heart's action (cardiac cycle) continues 0.9 sec, of which atrial contraction (atrial systole) takes from 0.1 to 0.12 sec, and ventricular contraction (ventricular systole) 0.3 sec (at this time the atria are already at rest). The interval between atrial contraction and ventricular contraction, which is equal to the period required for excitation to be conveyed through the conducting system ranges from 0.06 to 0.1 sec. The third phase of the cardiac cycle—the pause, diastole of the entire heart—continues 0.4 sec.

During the atrial systole, the blood is supplied from the right atrium to the right ventricle, and from the left atrium to the left ventricle. During the ventricular systole, the blood is conveyed from the right ventricle through the pulmonary artery into the pulmonary circulation, and from the left ventricle into the aorta and further into the vessels of the systemic circulation. The blood from the pulmonary circulation is delivered into the left atrium, and from the venae cava inferior and superior into the right atrium.

During a diastole, the tricuspid and bicuspid valves open into the ventricular chambers, and by this the blood is supplied into the cardiac cavity, while the semilunar valves of the pulmonary artery and the aorta are closed, preventing the backflow of blood from the pulmonary circulation into the right ventricle, and from the aorta into the left ventricle. During a contraction of the heart, other additional impulses are unable to provoke its excitation, since in this period the heart is refractory, i.e., it does not respond to new stimuli.

An impulse generated in the sinoatrial node and in other points of the conducting system is considered automatic, but its automatism is governed by the vagus nerve and the sympathetic nerve, which innervate the heart. Stimulation of the vagus nerve decelerates the rhythm of the heart's action, whereas stimulation of the sympathetic nerve produces the opposite effect. On the aorta, the pulmonary artery and the coronary arteries of the heart, the vagus nerve and the sympathetic nerve form plexuses ending in aggregations of nerve cells—ganglia, from which nerve fibres branch off to innervate the muscle fibres. The activity of the parasympathetic (vagus nerve) and the sympathetic nervous systems is regulated by the cerebral cortex.

In addition to the above-mentioned two nerves, I. P. Pavlov discovered another nerve, which intensifies cardiac systole by a trophic influence on the heart muscle, i.e., by improving the nutrition of the muscle and mobilizing its vital capacity.

During a systole, 50-60 ml of blood is ejected from the left ventricle into the aorta, which is known as the stroke volume. The blood is pumped through the arteries of the general circulation by the force of cardiac contraction as well as by dilatation of the elastic vessels during a systole and their constriction during a diastole. The arterial blood flows through the arteries under pressure, which in the brachial artery is equal to 120-80 mm Hg, i.e., 120 mm during a systole (systolic pressure) and 80 mm during a diastole (diastolic pressure). Therefore, the maximal arterial pressure is also called systolic, and the minimal one, diastolic. The venous pressure in the ulnar vein does not normally exceed 7 mm Hg, which corresponds to 100 mm of water. It is known that under normal conditions, notwithstanding the various factors working to decrease or heighten the arterial pressure, the latter constantly remains at the same level. Owing to I. Pavlov's discoveries it has been possible to establish that the stability of pressure is ensured by the receptors embedded in the vessel walls, which balance it by a reflex reaction. The capillary vessels are sharply dilated on physical exertion. This, however, does not entail a decline in the general pressure, since in this case the reflex stimuli from the carotid sinus will cause evacuation of the blood depot, constriction of the arterioles and an increase in the amount of circulating blood. It should be noted that normally not all of the blood circulates in the vascular bed. Of the 5-6 litres of blood contained in the human body, one-third is at rest in the so-called blood depots—the spleen, the liver, the subpapillary muscles of the skin and the intestinal veins.

The blood flow through the vascular network is irregular: in the initial parts of the systemic circulation—the arteries—it is fast, and is considerably decelerated in the capillaries. After the blood has passed from the arterial system into the venous network, its movement toward the right heart occurs under the action of new factors. The most important of them is the suctorial effect of the heart and the thoracic cavity. Within the latter, the pressure is negative, i.e., lower than the atmospheric pressure, whereas in the veins the pressure is positive. As a result of this difference in pressure the blood flows where the pressure is negative. The flow of venous blood toward the right heart is especially increased during inspiration, because at this time the pressure in the thoracic cavity falls to an even lower level. The blood current from the periphery to the heart is accelerated by the contractions of skeletal muscles which push blood through the veins in the direction of the heart. The blood flow towards the heart is facilitated also by the work of the diaphragm, which during inspiration presses down upon the liver and squeezes blood out of it into the inferior vena cava.

SYMPTOMS OF CARDIOVASCULAR DISEASES

Dyspnoea. This is one of the main and earliest complaints in heart disease. In mild cases, it troubles the patient only on physical exertion, in moderately severe cases, during performance of usual work, and in cases of far-advanced changes in the heart, it appears even at rest. The onset of dyspnoea is accounted for, firstly, by stasis in the pulmonary circulation and irritation of the endings of the vagus nerve in the lungs, secondly, by a disturbance in the cerebral circulation and irritation of the medulla oblongata by inadequately oxygenated blood (hypoxaemia). In pulmonary emphysema and pulmonary fibrosis causing cardiac insufficiency, the respiratory surface and vital capacity of the lungs are decreased. The attendant quickened respiration and its shallowness make the conditions of arterialization of the blood still more unfavourable. Dyspnoea is a distressing symptom also because the quickened respiratory movements increase the load on the heart.

Tachycardia. Tachycardia is most commonly a sensation of an excessive rapidity of the heart's action. It may be one of the early signs of cardiac insufficiency, appearing on insignificant physical exertion, after a hearty meal or emotional strain. In neurosis, intoxication, and anaemic conditions tachycardia is due to a heightened excitability of the heart. Tachycardia may also be noted in persons with a normal heart in various emotional states. Sometimes a patient complains of tachycardia in the presence of a normal pulse rate, and, conversely, in spite of a quickened pulse rate tachycardia may be absent.

Pain. Pains in the heart region sometimes occur in persons with a normal heart as a result of increased excitability of the nervous system. More frequently, however, they are important subjective symptoms of a cardiac disease. Pains due to thrombosis, atherosclerotic constriction or a functional spasm of the coronary vessels are known as anginose pains. In such cases acute anaemia of the myocardium develops which is attended by compressing retrosternal pains, now burning, now dull in character, radiating to the left arm, shoulder-blade, neck, etc. In acute pericarditis, pains are continuous. Dull retrosternal pains unconnected with physical exertion occur in syphilitic aortitis as well as in sclerosis of the aorta. These pains are apparently caused by inflammatory changes and possibly by compression of the nerve plexuses of the outer coat of the aorta. They are continuous and, as distinct from anginose pains, cannot be relieved by vasodilating drugs.

Dizziness. Dizziness, headaches, fainting, and debility often occur in patients with cardiovascular diseases.

Fever. Fever in a heart patient may be due to an inflammatory process in the heart and the resultant complication. It is found in endocarditis, pericarditis, as well as in myocardial infarction, pulmonary infarction, and thrombophlebitis.

It is necessary to note that in patients with a heart disease concomitant inflammatory conditions, including lobular pneumonia, may take place with an insignificantly elevated temperature.

Blood-spitting. In heart diseases, blood-spitting is caused by stasis in the pulmonary circulation and pulmonary infarction. It manifests itself by separate spits or a massive haemorrhage, as it sometimes occurs in stenosis of the mitral opening. Very rarely, a massive pulmonary haemorrhage ending in death develops from a rupture of an aortic aneurysm into the air passages.

Findings on inspection. The position of a patient with a heart disease depends on the condition of the heart. Patients with mild forms of cardiac insufficiency prefer to lie on the right side since lying on the left side involves distressing sensations. In insufficiency of the left ventricle, the patient prefers a sitting posture. Apparently, in this position the blood flow in the pulmonary circulation is facilitated by a greater influx of blood into the left atrium from the pulmonary veins. Patients suffering from continuous dyspnoea prefer to sit with their legs down; the legs in this position become considerably swollen but dyspnoea is less distressing. When stasis extends to the venae cava and the liver the patient may lie down.

Patients with vascular insufficiency feel better only in a recumbent position which facilitates supply of blood to the brain.

In a number of cardiovascular diseases, there is a distinctive skin pallor, which may be due to a spasm of the skin vessels, anaemia, intoxication, or emptiness of the peripheral vessels. A marked pallor of the skin is noted in insufficiency of the aortic valves, in collapse, as well as in severe rheumatic heart disease. In lingering septic endocarditis the skin takes a characteristic colour of coffee with milk.

In heart diseases, the skin is cyanotic, mainly in the peripheral parts of the body: the extremities, the face, the lips, the tip of the nose, and the ear lobes. Therefore, cyanosis in heart disease is most often called *acrocyanosis* (cyanosis of the extremities). In severe cardiovascular insufficiency, acrocyanosis develops into general cyanosis.

With a decelerated blood flow there is an increase in heat release into the atmospheric air, so the peripheral parts of the body (hands, feet) become cold, which is felt by the patient himself and is determinable by objective examination—palpation and measurement of the skin temperature. A cyanotic discoloration of the skin is also noted in persons with a pulmonary disease interfering with arterialization of the blood; in such cases, however, the extremities are warm, since the peripheral blood flow is not decelerated, and the heat release into the atmospheric air is not increased.

In heart diseases oedema appears with the onset of cardiovascular insufficiency and may be localized in the subcutaneous areolar tissue (anasarca), the pleural cavity (hydrothorax), the abdominal cavity (ascitis), etc. The site of oedema depends on the position of the patient: in ambulant patients, it is confined to the ankles, in bed-ridden patients, to the lumbar and sacral region. In the event of a considerable oedema the fluid may spread throughout the body. Oedema is due to several causes. The most important of them is an increased pressure in the venous capillaries. While the blood from the arterial limb is intensively penetrating into the tissues, its reverse absorption from the tissues into the vascular bed is disturbed as a result of an increased pressure in the venous limb of the capillaries. The venous pressure is the highest in the veins of the lower extremities; therefore, cardiac oedema in ambulant patients is localized above all in the feet and shins. Accumulation of fluid in cardiac oedema is also promoted by the following factors: (a) inadequate excretion by the kidneys of common salt which is readily deposited in the tissues; (b) an increase in the reverse absorption of water by the renal tubules. In the late stages of cardiac insufficiency, oedema also grows partly because of protein deficit in the blood plasma and a decrease in the oncotic pressure of the blood. The onset of oedema is due in large measure to a disturbance in the permeability of vessels which, due to altered conditions of nutrition, let fluid pass into tissues.

Changes in the extent of oedema are determined by measurement of the daily liquid intake and urinary discharge as well as by regular measurement of the body weight. Liquids are eliminated from the body mainly through the kidneys and partly through the lungs, faeces and skin; therefore, the patient's weight is the most accurate index of water metabolism.

EXAMINATION OF THE HEART AND BLOOD VESSELS

Inspection of the Precordium and Vessels

Under normal conditions, inspection of the chest shows a rhythmic protrusion and retraction of its wall in the fifth intercostal space, somewhat inwardly to the midclavicular line. This is caused by systolic contractions of the heart and is known as the apex beat, i.e., the stroke of the heart apex against the chest wall. In heart disease, the apex beat becomes diffuse, and in this case a stroke from contraction of the front wall of the heart is seen to the left of the sternum, as well as cardiac pulsations in the superior part of the epigastrium. Sometimes, inspection of the precordium reveals a circumscribed protrusion—the so-called precordial bulge which develops from deformation of the thorax due to pressure by an enlarged heart in the period of its development.

Inspection of the vessels yields some valuable findings. On dilation of the aorta, pulsation is visible at left and at right in the second intercostal space. A pulsating protrusion is also found sometimes. Pulsation of the carotid, subclavian and the more superficial brachial, radial, and temporal arteries may also become visible under certain conditions. The examiner should have in mind a possible tortuousness of the vessels, which points to induration of their walls (sclerosis). On examination the veins may be dilated and congested as a result of general venous stasis as well as a local disturbance in venous blood drainage. Compression of a vein by a tumour, a scar or its obstruction by a thrombus is followed by oedema developing about the vein, while the venous collaterals become dilated. Mediastinal tumours cause facial oedema and dilation of the veins of the chest, neck, and upper extremities. Thrombosis of the portal vein leads to dilation of the collaterals around the navel, forming the so-called caput medusae. General venous stasis is found with diseases of the heart. In such cases, there is considerable bulging of the external jugular veins on the neck, which is more pronounced on the right side. In insufficiency of the right ventricle, their systolic pulsation is also notable, whereas normally the jugular veins collapse during a systole.

Sometimes, finger pressure on a nail, strong enough to leave a white spot in the middle, may show the appearance of redness simultaneous with cardiac pulsation, followed by paleness. The same phenomenon is noted after rubbing the forehead to redness with a towel or handkerchief. In both cases, there is pulsation of the capillaries—the capillary pulse which is encountered in aortic valve insufficiency.

Sometimes, one may note a circumscribed dark-red dilatation of the minute terminal peripheral vessels of the head, face, and extremities, which is known as telangiectasis.

Palpation of the Heart Region

Palpation of the heart region may give valuable information on the character of the heart's action and changes in the heart muscle. During a systole, the heart apex is displaced forward and upwards, and is pressed against the chest wall, producing the sensation of a stroke by a resilient dense object. For detecting the apex beat the hand is placed palm down on the heart region to find the site of the beat and after this its height and strength are determined with the index finger. The apex beat is undetectable when the stroke is against a rib or when the intercostal spaces are covered with a thick layer of adipose tissue. In case of fluid collection in the pericardial cavity, in pulmonary emphysema, etc., the apex beat is undeterminable because of the heart being pressed back from the frontal chest wall. In certain cases, it gives a sensation of elevation and resistance, which is determined by finger pressure on the site of the apex beat. An elevating stroke is found in all cases of hypertrophy of the left ventricle. When the hypertrophied heart muscle is weakened, the apex beat is lessened in strength and becomes diffuse.

The location of the apex beat may help determine the left heart boundary. With a considerable hypertrophy of the left ventricle, the apex beat is displaced not only leftwards but also downwards. The largest displacement of the apex beat to the left and downwards is seen in aortic valve insufficiency and hypertensive vascular disease. In these cases, it is found in the sixth and even in the seventh intercostal space, several centimetres outwards from the left midclavicular line. Displacement of the apex beat is also observed in dislocation of the heart to the left in right-side wet pleurisy, pregnancy, ascitis, meteorism, and adhesive processes in the left lung. Palpation of the epigastrium may help reveal pulsation which occurs in hypertrophy and dilatation of the right ventricle. The friction sound felt by the hand in the heart region is notable in pericarditis. Adhesion of the pericardium to the chest wall causes retraction at the site of the heart apex rather than protrusion of the chest wall during a systole. This points to a negative or paradoxical apex beat. On palpation of the heart region, one may sometimes feel vibration or thrill of the chest wall, which is known as a purring thrill. It is encountered during a diastole in constriction of the left venous orifice, is felt at the site of the apex beat and has a low timbre. A systolic vibration of the chest wall is also notable in stenosis of the mouth of the aorta or pulmonary artery, and is felt by the palm applied to the chest in the region of the heart base.

Percussion of the Heart

Determination of the heart boundaries by percussion is based on that fact that on percussion a lung containing air gives out a clear pulmonary sound, whereas airless organs, the heart in particular, produce a dull sound.

In the places where the heart is covered by the lungs, the sound elicited on percussion is dull and tympanitic, and where the heart is not covered, it is dull.

The heart's dullness may be relative or absolute (Fig. 35).

The area of dullness corresponding to the part of the heart immediately adjoining the chest wall is called absolute dullness; the region of a muffled percussion note corresponding to the true size of the heart is known as relative dullness.

Absolute dullness may change depending on a number of circumstances. It is increased in dilatation of the heart when the pulmonary margins are displaced and in case of fluid accumulating in the pericardium; the absolute dullness is decreased in pulmonary emphysema when the distended lung margins overlap the heart and cover almost the whole of it.

The borders of relative cardiac dullness. Percussion may be performed by striking a finger with a finger or a pleximeter with a plexor,

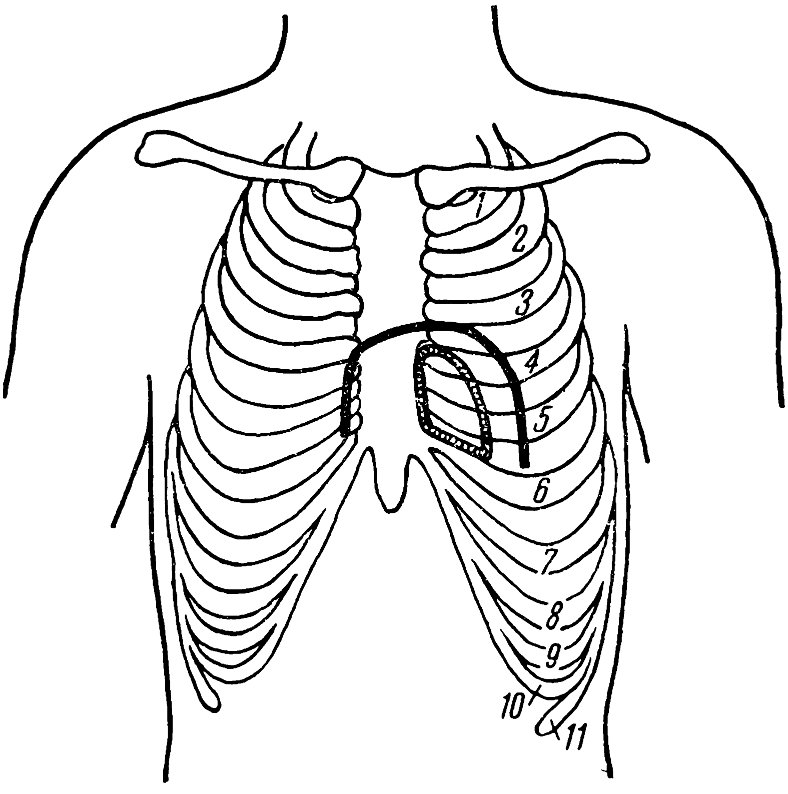


Fig. 35. Border of absolute and relative cardiac dullness (1st-2nd ribs)

with a moderate effort. In finger percussion, the finger used as a pleximeter should be firmly applied to the chest parallel to the border being searched for. The first step is to determine the relative dullness of the liver along the right midclavicular line, after which percussion is continued towards the sternum along the intercostal space lying immediately above the relative dullness of the liver, whereupon the right border of the heart is found. In order to determine the left border of the heart, the apex beat is preliminarily found by palpation, after which percussion is continued in the direction of the apex beat, starting from the armpit, along the intercostal space, until the apperance of a dull sound. As a rule, the area of dullness coincides with the site of the apex beat. The upper border of the heart is determined by percussion from above downwards along the left sternal line.

Normally, the right border of the heart lies 1 cm outwardly to the right margin of the sternum; the left border, along the left midclavicular line or 1 cm inwardly to it; the upper border, in the third intercostal space.

The borders of absolute cardiac dullness. The area of absolute cardiac dullness is determined by slight percussion in the direction from the clear to the dull pulmonary sound, or from the site of the greatest dullness to that of the clear sound. Absolute cardiac dullness is not "femoral" in character because the position of the lungs, the stomach and the intestine in proximity to the heart lends it a tympanitic quality. The right border of absolute dullness normally begins from the lower margin of the 4th costal cartilage along the left margin of the sternum. The upper border lies on the 4th rib, and the left—1.5 cm inwardly to the midclavicular line.

The width of the vascular bundle is determined by percussion in the second intercostal space, and is equal to 5-6 cm (Fig. 35).

Changes in the borders of cardiac dullness. In hypertrophy and dilatation of the left ventricle the heart is mainly enlarged to the left, and sometimes to the left and downwards, reaching the sixth or seventh intercostal space. With an enlarged left atrium, the area of absolute dullness extends to the 3rd rib, and that of relative dullness, to the 2nd rib.

In chronic diseases of the lungs involving formation of scar tissue and a reduction in the respiratory surface the heart is enlarged to the right; the same change is notable in stenosis of the left atrioventricular orifice.

In mitral defects and hypertensive vascular disease in the stage of circulatory disorder, the heart is enlarged in all of its parts and in every direction—to the right, to the left and upwards; this condition is known as the ox heart (cor bovinum). During shrinkage processes in the lungs and the pleura, the heart is sometimes pulled to the affected side; with an increased pressure in one half of the thorax (wet pleurisy, pneumothorax) the heart is displaced toward the normal side. The heart may be located in the right half of the thorax as a result of abnormal development.

Auscultation of the Heart

Judgement on the heart's action may be formed from cardiac sounds, for which purpose the heart is auscultated in order to determine their intensity, rhythm and clearness. Sounds given out by the heart are heard directly with the ear as well as with the aid of a stethoscope or phonendoscope. Under normal conditions, two cardiac sounds are audible. The first sound is otherwise called systolic sound, since it occurs during a systole and is due to heart muscle strain and closure of the bicuspid and tricuspid valves. The first sound is best heard at the apex of the heart, and is lower in timbre than the second sound. The second, or diastolic, sound is given out at the beginning of a diastole by vibrations of the valves of the aorta and the pulmonary artery and the backflow of blood from the aorta and the pulmonary artery. It differs in timbre from the first sound in being shorter and softer.

The first sound is separated from the second by a brief pause which corresponds to the period of ventricular systole. The second sound is separated from the first by a long pause corresponding to the diastolic period.

In affection of the valve apparatus and stenosis of the orifices through which blood enters the heart and is pumped out into the vascular system, murmurs are heard in addition to cardiac sounds. In case of a total or partial destruction of the heart valves, the blood during a systole or diastole flows also in the reverse direction (regurgitation), which is responsible for cardiac murmurs. A murmur instead of a sound also occurs when the openings between the atria and ventricles or between the ventricles and the aorta and the pulmonary artery are narrowed in consequence of an inflammatory process in the valves, vascular atheromatosis or congenital defects.

The sites of hearing cardiac sounds and murmurs. Cardiac sounds and murmurs are best heard where they are best conducted. These localities do not correspond to the projection of the site of their origin onto the chest, but lie at a certain distance away (Fig. 36).

Auscultation of the heart is performed according to plan. At first, one has to auscultate the mitral valve, then the aortic valves, the valves of the pulmonary artery, and the tricuspid valve.

The sounds and murmurs originating at the site of the bicuspid valve guarding the left atrioventricular orifice are heard at the apex of the heart.

The sounds and murmurs originating in the region of the valves guarding the aortic orifices are best heard at the right sternal margin in the second intercostal space. Those arising in the place of attachment of the valves of the pulmonary artery are audible in the second intercostal space to the left of the left sternal margin.

For hearing the diastolic murmur occurring in aortic valve insufficiency there is a fifth, Botkin's point located in the third intercostal space to the left of the sternum. The sounds and murmurs arising at the site of the tricuspid valve guarding the right atrioventricular orifice are audible at the lower end of the sternum at right.

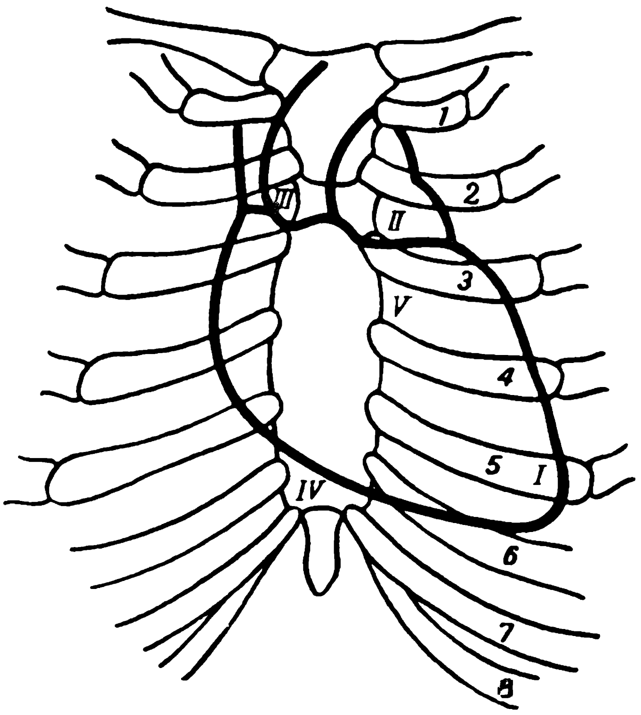


Fig. 36. Sites of auscultation of the valves

I—mitral valve; *II*—valves of the pulmonary artery; *III*—aortic valve; *IV*—tricuspid valve; *V*—Botkin's point; *1-8* — ribs

Changes in cardiac sounds. In auscultation of the heart in a healthy person, the first sound at the heart apex is louder and longer than the second sound which is shorter and is less sonorous. Over the heart base the second sound is louder than the first and has uniform sonority both over the pulmonary artery and the aorta. A decrease in sound intensity is notable with a thickened chest wall, fluid collection in the pleural cavity and in the pericardial cavity, which causes displacement of the heart from the chest wall, as well as with distension of the lungs with which the heart is covered.

The cardiac sounds are louder in persons with thin chest walls, during shrinkage processes occurring in the lungs, in excitement, and as a result of a reduced viscosity of the blood in anaemia.

A change in cardiac sounds is also noted in heart disease. With an increased heart's action, the first sound is, as a rule, more sonorous, and with a weakened heart muscle it becomes muffled. A weakening of the first sound is noted in incompetence of the heart valves, endocarditis, and acute myocarditis. For a sound to be sonorous very important is not only the contractile capacity of the muscles but also the quickness of contraction and the degree in which the ventricles are filled with blood before a contraction. The quicker the contraction of the heart muscle the more sonorous the first sound, which also becomes sonorous when heart contractions in the presence of strong ventricular muscles take place with inadequate ventricular filling. Therefore, an intensification of the first sound is not always a sign of a good heart's action. In hypertrophy of the heart muscle, when the heart contractions are slower, the first sound at the heart apex is weakened, whereas in stenosis of the mitral opening it is intensified. Nevertheless, the condition of the heart muscle is better in hypertrophy than in stenosis of the mitral opening.

With a heightened arterial pressure (hypertensive vascular disease, chronic nephritis, etc.), the second sound on the aorta is intensified. This is known as accentuation of the second sound over the aorta. A weakening of the second sound over the aorta is found in incompetence of the aortic valves as a result of induration and partial or total destruction. An intensified second sound over the pulmonary artery is noted with a heightened pressure in the pulmonary circulation and the resulting closure of the valves of the pulmonary artery under a great pressure in the diastolic phase (in mitral disease and pulmonary diseases involving the capillary network of the pulmonary circulation).

Duplication of heart sounds. Duplication of the first sound is in the majority of cases associated with a change in the heart muscle. It may be due to splitting of the valvular and muscular components of the first sound, asynchronous contraction of the right and left ventricles, and premature contraction of the atria. In a certain percentage of cases, duplication of the first sound is encountered in healthy persons after physical strain.

Duplication of the second sound is due to an asynchronous closure of the aortic valves and the valves of the pulmonary artery.

The less the diastolic blood volume in a given ventricle, the shorter the systole and the earlier the closure of the valves of the corresponding vessel. For example, in mitral stenosis the aortic valves close earlier than those of the pulmonary artery, therefore, the usually fused second sound will be heard as duplicated over the pulmonary artery (duplication of the second sound, or “quail rhythm”).

The gallop rhythm. The gallop rhythm occurs in cases where splitting of the first sound is markedly audible being perceived as two independent sounds, with the appearance of a triplet rhythm. The additional third sound may be presystolic when it is heard nearer the first sound; systolic, when arising immediately after the first sound; and diastolic, when heard in the middle of a diastole.

The presystolic additional sound takes its origin from separation of the atrial and ventricular components of the first sound.

The systolic gallop rhythm comes on the stage as a result of a delay in contraction of one ventricle compared with the other. The origin of the additional diastolic sound is uncertain.

The additional sound is duller and shorter than the two principal sounds, and is determinable only in tachycardia. It is highly important to know that the appearance of gallop rhythm is a sign of a severe disturbance in the heart’s action; for this reason, many call the gallop rhythm “the heart’s appeal for help”.

Cardiac murmurs. Cardiac murmurs are subdivided into organic and functional.

The functional or nonorganic murmurs are not those associated with organic lesions of the valve apparatus or the heart muscle but those caused by a functional disturbance of its nerve apparatus as well as by a change in the blood flow or composition of the blood (accelerated blood current, decreased viscosity of the blood).

Organic murmurs occur in connection with inadequate valve closure (valve incompetence and constriction of the atrioventricular openings, stenosis of the orifice of the aorta and the pulmonary artery).

The blood current within the heart does not produce any murmur. However, the passage of blood through narrowed orifices from one cardiac chamber into another or from the heart into a large vessel gives rise to turbulence of the flow and vibrations of the chamber walls which are heard as murmur on auscultation. The intensity of the murmur depends on the strength of cardiac contractions and the degree of stenosis. A murmur may cease altogether in considerable constriction of the orifices and a marked weakening of the heart muscle. Murmurs occurring during a ventricular contraction are termed systolic and those in the phase of diastole, diastolic. Murmurs may also arise from extracardial causes, for example, from changes in the pericardium or the pleura adjoining it, and changes in the larger vessels. As to their timbre murmurs may be blowing, harsh, musical.

A systolic murmur is clearly audible at the heart apex and may result from incompetence of the mitral valve. Normally, in the period of cardiac systole the bicuspid valve completely closes the left atrioventricular orifice so as to prevent the backflow of blood into the atrium. In mitral valve deformity, however, the left atrioventricular orifice is partly left open, and in the systolic phase the blood from the ventricle is forced back under pressure through a narrow fissure into the left atrium. This is accompanied by a murmur isochronous with the systole and therefore called systolic. It also occurs with an intact mitral valve, but when the left ventricle is considerably enlarged.

In this case, there is an expansion of the fibrous ring and, consequently, of the left atrioventricular orifice, to a degree where the valve is unable to close the opening completely. This is known as relative incompetence of the mitral valve.

A systolic murmur may arise also in the absence of relative incompetence of the mitral valve, but with a weakened heart muscle. In such cases, it is identified as muscular-functional murmur.

The mechanism of origination of the systolic murmur audible over the lower part of the sternum in affection of the tricuspid valve is the same as that producing analogous murmur at the heart apex. A systolic murmur over the aorta and the pulmonary artery is produced by the passage of blood through a narrowed orifice into the vessel under a large pressure.

Anaemic (haemic) murmurs are audible all over the cardiac region, but are heard best over the pulmonary artery and the aorta.

The diastolic murmur at the heart apex appears in stenosis of the left atrioventricular orifice and deformation of the mitral valve. The blood passing through the constricted orifice from the left atrium into the left ventricle gives rise to a murmur which is well audible at the beginning of the diastolic phase, weakens in the middle of a diastole and becomes the most sonorous at the end of a diastole. The murmur heard at the end of a diastole and the beginning of a systole is termed presystolic murmur. The diastolic murmur heard over the second right intercostal space or in Botkin's point is produced by the backflow of blood from the aorta into the left ventricle in aortic valve insufficiency.

Pericardial friction sound. Normally, friction of the pericardial surfaces produces no sound at all. In the presence of inflammation, fibrin is deposited on the pericardial surfaces which become rough, and in the period of cardiac contraction a rub is heard both during the systole and diastole. The friction sound may be faint, but often it becomes coarse and scraping, and sometimes can be felt by the palm applied to the heart region.

Examination of the Pulse

Examination of the pulse, determination of its frequency, rhythm and quality is an important method of ascertaining the state of the heart's action.

The pulse is palpated in the radial artery at the wrist with three fingers: the index, the middle and the fourth, with which the artery is slightly pressed to the radius. The pulse should preferably be taken from both hands so as to find out a difference, if any.

The pulse wave is formed as a result of the artery being filled with blood ejected from the aorta; therefore, the frequency and speed of pulse waves, and the degree of filling of the radial artery are directly conditional on the state of the heart.

The pulse is examined for the following five characteristics: (a) frequency; (b) rhythm; (c) volume; (d) tension; (e) rate. In a healthy person the number of pulse waves ranges from 70 to 80 beats per minute. In physical exertion as well as in emotional strain, the pulse is quickened. Frequent pulse is noted in circulatory disorders and in myocardial dysfunction. A quickened pulse is also observed in intoxications, metabolic disorders, hyperfunction of the thyroid gland, fever, and increased nervous excitability. In the latter case, the pulse is termed labile, i.e., unstable.

A deficient pulse (pulsus deficiens) is present in cases where not all cardiac contractions reach the radial artery, which is evidence of a disturbance in the cardiac rhythm.

The pulse shows characteristic variations in volume: it may be full with a normal heart muscle, and faint, or incomplete, in myocardial weakness when the amount of blood delivered into the aorta is

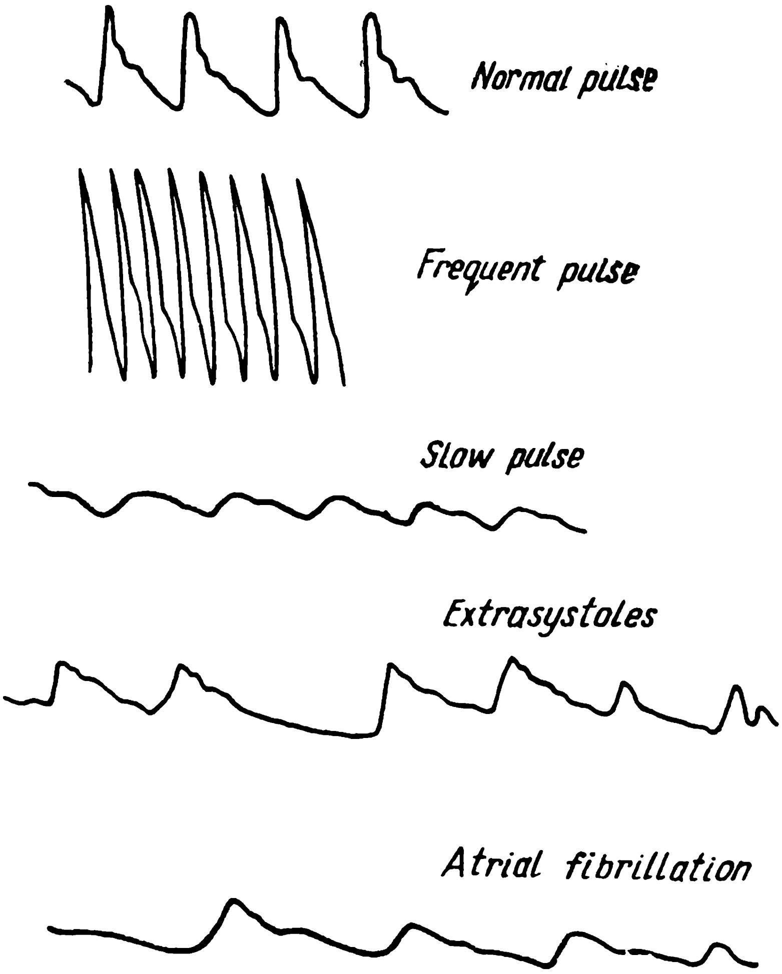


Fig. 37. Pulse curves

reduced. One should also give attention to the pulse tension, which is determined from the force of pressure on the radial artery required to stop the pulse wave. When it is necessary to use considerable force, a hard pulse (pulsus durum) is present, which occurs with an increased arterial pressure. Disappearance of the pulse upon a slight pressure applied to the radial artery is noted in a decreased arterial pressure; such a pulse is called soft (pulsus mollis). With a considerable weakening of cardiac function and a low arterial pressure, the pulse becomes thready (pulsus filiformis).

With regard to its rate, the pulse may be quick (pulsus celer), i.e., distinguished by a quick rise of the pulse wave followed by its just as rapid decline. This pulse is noted in aortic valve insufficiency. The pulse may also be slow (pulsus tardus); this pulse is present in stenosis of the aortic orifice and hypertension. In examining the pulse, attention should also be paid to the condition of the arterial wall, which depends on the elastic and muscle tissues it contains. In atherosclerosis, as a result of proliferation of connective tissue in the vessel walls and deposition of lime salts within the latter, the arteries become thickened, less compressible and roll under the finger like a cord. Different pulse (pulsus differens), i.e., the absence of the pulse on one hand or disparity between the pulse volumes on both hands, is an important finding if only this difference is not the result of an abnormal development and position of the radial artery. In pathology, a different pulse is found with aortic aneurysms and in compression of the brachial artery by a tumour, enlarged lymphatic glands, etc.

Examination of the pulse is carried out not only by finger palpation but also with the aid of a sphygmograph. Fastened to the forearm with a special contrivance this instrument records the features of the pulse. The recording is made on a smoked chart wound on a rotating reel. The tracing thus obtained is called a sphygmogram (Fig. 37).

Measurement of the Arterial Blood Pressure

For measurement of the arterial blood pressure (Fig. 38), the upper arm is compressed with a special air-filled cuff connected to a mercury or spring-type manometer with a scale graduated in mm Hg. At the same time, the pulse is felt with the fingers. The cuff is inflated for some time after the palpable pulse disappears. Then the air is gradually let out of the cuff. The point at which the pulse reappears corresponds to the systolic pressure. Complete disappearance of the pulse corresponds to the systolic pressure. This method, however, is defective in that it fails to determine the diastolic pressure which is highly important in clinical practice. The auscultatory method proposed by N. S. Korotkov makes it possible to determine not only the systolic but also the diastolic pressure. The procedure is as follows: A phonendoscope or stethoscope is pressed lightly below the cuff medially in the antecubital fossa, where the ulnar artery can be felt. When air is pumped into the cuff, the artery is compressed and no sound is heard over the artery. As the cuff is deflated, the moment sounds first become audible corresponds to the systolic pressure. As deflation proceeds, the sounds become at first louder,

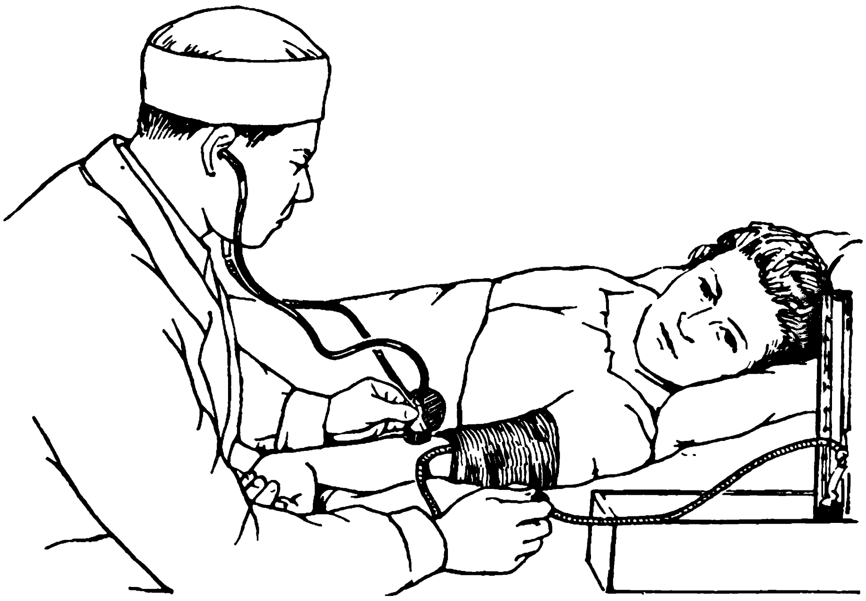


Fig. 38. Measurement of the arterial pressure

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then muffled, and then disappear completely. The moment of sound disappearance corresponds to the diastolic pressure. Besides mercury manometers, the arterial pressure is also measured by means of spring-type manometers in which pressure is indicated by a pointer. The moment at which the pointer stops fluctuating corresponds to the maximal pressure, and the moment of the least fluctuations, to the minimal pressure. Measurement of the arterial pressure by means of a spring manometer may be combined with auscultation of the ulnar artery by the method suggested by Korotkov.

The systolic arterial pressure is isochronous with the left ventricular systole. The interarterial pressure during a diastole falls to a very low level. The diastolic pressure depends, on the one hand, on the blood volume in the vessels and, on the other hand, on their tone. Normally, the systolic arterial pressure ranges from 120 to 140 mm Hg, and the diastolic pressure, from 60 to 80 mm Hg.

The difference between the systolic and the diastolic pressure is known as the pulse pressure. Normally the pulse pressure is equal to 40-60 mm Hg. The arterial blood pressure is subject to insignificant diurnal variations, rising on physical exertion, excitement, mental work, after meals, and falling after a rest. The arterial pressure tends to increase with advancement in years, and in old age may again return to normal.

RHEUMATIC FEVER (RHEUMATISMUS)

Rheumatic fever is a systemic disease of connective tissue (mesenchyma) of infectious-allergic origin and a frequent cause of disorders in the cardiovascular system. As a rule, rheumatic fever begins in childhood and adolescence and early produces disablement. Formerly, rheumatic fever was listed among articular affections; however, the French physician Bouillaud and the Russian clinician G. I. Sokolsky almost at the same time (1835-36) and independently of one another, isolated rheumatic fever from the group of articular affections as a separate malady and pointed to an inevitable involvement of the heart in this disease. There is a meaningful aphorism: "Rheumatic fever licks the joints, even the meninges, but bites the heart."

Terminology. Each country has its own terminology for rheumatic fever. In the Soviet Union, it is customarily called rheumatism or Sokolsky-Bouillaud disease. The affection by this disease of the cardiovascular system and sometimes of the joints is considered inevitable. All other affections of the joints of an infectious-allergic origin are listed among nonspecific or rheumatoid arthritis, i.e., resembling rheumatism. In the United States, this disease is known as rheumatic fever, in the Netherlands, as infectious polyarthritis.

Aetiology and pathogenesis. Rheumatic fever often develops after a streptococcic (β-streptococci of group B) focal infection (tonsillitis, erysipelas, scarlet fever, etc.). Focal infection becomes responsible for a person's hypersensitivity to heterogenous protein—antigen, as well as to such unfavourable factors as cold, catarrhal conditions, etc. In the foci of rheumatic inflammation, however, such as in the heart, the vessels, the joints, etc., streptococci are absent, but the patient's blood contains antistreptococcic, otherwise called immune, bodies which are evidence of an active pathologic process. The mechanism of their formation is supposed to be as follows. The collagen tissue contains hyaluronic and chondroitinsulphuric acids which seem to cement this tissue and make it impermeable. In rheumatic fever they are released and broken up. Hyaluronic acid breaks up under the influence of hyaluronidase whose formation is connected with streptococci. That is why it is otherwise termed streptohyaluronidase. In response to the formation of streptohyaluronidase, antihyaluronidase is usually formed in the body. Consequently, the presence in the blood of an excessive amount of antihyaluronidase is one of the indices of the activity of the rheumatic process. Streptococci also produce a substance that lyses erythrocytes (streptolysin) as well as substances capable of dissolving fibrin (fibrinolysins). Both streptolysins and fibrinolysins give origin to antibodies forming in the blood, i.e., antistreptolysin and antifibrinolysin. An increase in the blood of antihyaluronidase above normal figures (300-350 units) as well as of antistreptolysin, whose normal level is within a range of 250-350 units points to an active rheumatic process.

Thus, the high titre of antihyaluronidase, antistreptolysin, and antifibrinolysins is a sign of an active rheumatic process taking place in the body. All of the above-mentioned antibodies as products of the body's own broken-up proteins turn into antigens, autoaggressins, in other words, substances which in the absence of streptococci may provoke another exacerbation of the rheumatic process.

Among the objective signs showing a heightened body reactivity are an elevated ESR, a positive reaction of C-reactive protein, and eosinophilia; they, however, are nonspecific, so immunologic reactions are more demonstrative.

In rheumatic fever, changes occur in the blood plasma proteins. There is a considerable increase in the amount of coursely dispersed proteins (fibrinogen, globulin) and a reduction in the level of albumins consisting of small particles of a low molecular weight. In the period of exacerbation of the disease there is an increase in the amount of α_2- and γ-globulins, and an increase in fibrinogen.

Among rheumatic lesions of the visceral organs the most important are those of the cardiovascular system. Rheumatic myocarditis is a frequent disease running a benign course in the form of a focal affection of the heart muscle, but it may, though in rare cases, become diffuse and malignant in character. In rheumatic myocarditis the cardiac sounds are muffled, gentle systolic murmur is heard over the heart apex, the pulse may be arrhythmic, with tachycardia, fever, moderate leucocytosis, and an elevated ESR. The electrocardiogram shows a disturbance in the conduction of impulses from the sino-atrial node. The intervals between peaks P and Q grow to more than 0.2 sec. There are often changes in the coronary arteries (coronaritis) which are manifested by stubborn heart pains persisting even after administration of validol and nitroglycerin. The x-ray, even in the absence of cardiac defects, shows a dilation of the heart. Later, the rheumatic process extends to the endocardium also (see "Rheumatic Endocarditis").

It has been mentioned above that rheumatic fever is a systemic disease of connective tissue and, therefore, the rheumatic process may involve not only the heart but organs of mesenchymatous origin also, i.e., the vessels, including the coronary vessels, which sometimes leads to myocardial infarction. The affection of the cerebral vessels manifests itself by transient pareses and meningoencephalitis. Rheumatic diseases of the lungs are also known to occur as pneumonia, and vasculitis in the ramifications of the pulmonary artery. Fairly frequent blood-spitting in persons with heart defects is due not only to stasis in the pulmonary circulation but also to active rheumatic vasculitis and interstitial pneumonia resulting from it. Rheumatic fever also affects the serous membranes of cavities, sometimes with the pleura, the pericardium, and the peritoneum simultaneously (polyserositis), especially in childhood and adolescence.

Pathologic anatomy. The specific element of rheumatic fever is granuloma described by L. Aschoff (1904) and later studied in detail by V. Talalayev (1923). The latter subdivided the development of granuloma into the following stages: (a) exudative-alternative, with disorganization of collagen fibres and formation of an amorphous protein mass; (b) proliferative, with the formation of granulomas; (c) sclerosis—formation of connective and scar tissue at the site of granuloma. The first stage lasts 1-1$^1/_2$ months, the second stage 2-2$^1/_2$ months, and the third stage 3 months. The duration of all developmental cycles of the rheumatic process is not longer than 6 months. Morphologic changes specific for rheumatic fever may occur in various organs and tissues which take their origin from the middle layer of the embryo (the heart, vessels, muscles, tendons, supporting connective tissue in the liver, lungs, larynx, pharynx, etc.). Consequently, rheumatic lesions may be found wherever there is mesenchyma.

The classification of rheumatic fever was adopted at the plenary session of the Antirheumatic Committee in 1956. It refers to the phase, clinico-anatomic characteristics of the lesion, the course, and functional condition of the blood circulation. This classification draws a distinction between active and inactive rheumatic fever (i.e., in the period of remission). With an active process, note should be taken of the presence of acute (primary) rheumatic carditis, i.e., one with no heart defect, or relapsing rheumatic carditis, which may have a heart defect. In active rheumatic fever with primary and relapsing rheumatic carditis it is necessary to take note of the presence of rheumatic lesions in other organs and tissues. In an inactive rheumatic process attention is paid to the presence of rheumatic cardiosclerosis or a heart defect as well as the sequels and residual symptoms of the above-mentioned extracardial lesions. With regard to its course, an active rheumatic process may be acute, subacute, continuously recidivating and latent (concealed). In rheumatic fever, both in the active and the inactive phase, the circulation of the blood has to be characterized as follows: (a) absent circulatory insufficiency; (b) 1st-degree insufficiency; (c) 2nd-degree insufficiency; and (d) 3rd-degree insufficiency.

The following is an example of a comprehensive diagnosis of rheumatic fever: rheumatic fever, active phase, relapsing rheumatic carditis, mitral disease, 2nd-degree circulatory failure, polyarthritis, acute course.

The Articular Form of Acute Rheumatic Fever

Rheumatic fever more commonly develops one or two weeks after recovery from tonsillitis, influenza, otitis, paranasal sinusitis, etc. The disease begins with a rise in temperature to 38-40°C, and is accompanied by a multiple inflammation of the joints involving the knees, ankles, wrists, elbows, and shoulders. The joints are swollen and very painful on movement. The skin over them is hyperaemic, hot to the touch and damp. There is heavy perspiration, especially at night and in the morning, debility and general malaise. The patient lies motionless, fearing the slightest movement because of sharp pains in the joints. In some cases, a rheumatic attack becomes so diffuse as to affect even the joints of the vertebrae, the jaws and the small joints of the lower and upper extremities.

In the course of the disease the inflammatory process extends to other joints, and sometimes returns to the earlier affected joints, for which reason this disease has been dubbed migratory rheumatic fever. The disease at first affects one or two joints, and then more and more joints become involved (5 to 8 and even more). The affection of each joint develops within several hours. A joint which shortly before appeared healthy and was painless swiftly becomes swollen, painful and stiff.

In the affected joints the synovial membrane is inflamed and their cavities are filled with serous fluid as well as a small quantity of fibrin. The periarticular tissues are also involved in the disease process, showing inflammation and swelling. On palpation of the joints the most tender are the sites of attachment of fibrous fascias and tendons to bones.

As a rule, symptoms in the joints—pain, swelling, redness—pass within the first 5-7 days of treatment with salicylates and hormones, although the duration of articular affection may vary. Sometimes, the inflammatory process continues for 2-4 weeks and longer, while slight pains may persist even for several months. In the period of the disease, exacerbations are likely, with a new rise in temperature as a result of which fever becomes undulating in character. Articular attacks may recur rather frequently, sometimes several times a year. Cases are also known of another attack occurring after many years. As a rule, rheumatic fever leaves no stable changes in the joints, i.e., no ankyloses or deformities follow it.

Differential diagnosis. For a diagnosis of rheumatic fever the following four factors are highly important: a precursory tonsillitis, the migratory character of inflammatory changes in the joints, affection of the cardiovascular system (defects, vasculitis), and effective salicylate therapy. Rheumatic polyarthritis is sometimes difficult to distinguish from rheumatoid polyarthritis. For a differential diagnosis, one has to bear in mind that rheumatoid polyarthritis is preceded by influenza, scarlet fever, dysentery, gonorrhoea, etc. Moreover, in this form of polyarthritis, only some of the joints rather than all of them are inflamed simultaneously. At the same time, the inflammation is not migratory in character. The process takes a chronic course, and the joints rather quickly develop stable anatomical changes with stiffness, ankyloses, and changes in the shape of the bones and joints. In order to distinguish between rheumatic fever and rheumatoid polyarthritis one may be guided by the latter being comparatively resistant to salicylate therapy.

Prognosis. Rheumatic fever polyarthritis runs a more severe course in younger patients. At a young age, the disease is often very prone to repeated attacks and ends in a heart defect which in time leads to a considerable circulatory disorder. Elderly persons are rarely affected by rheumatism, and if they are, the disease takes a benign course. The heart defects so caused are compensated and involve no danger to life if physical and mental strain is somewhat limited.

Cutaneous Rheumatism

Rheumatic fever may also affect the skin in which case the capillaries are always changed. Because of an increased permeability of the latter small haemorrhages, the size of a pin-head, called petechiae, sometimes appear on the skin of the lower extremities (purpura rheumatica), more often on the extensor aspects of the joints. There is also an eruption of dense, painless nodules ranging in size from a millet to a pea (noduli rheumatici). Cases are also known of formation on the skin of the shin or the outer surface of the forearm of painful dark-red and dense nodules the size of a cherry, which are slightly elevated over the skin and penetrate its entire depth. This is the so-called nodular erythema (erythema nodosum) producing an eruption of nut-shaped nodules on the skin of the skull.

Rheumatic fever with predominant affection of the central nervous system. Rheumatic lesions may be found in the meninges, brain matter, and subcortical formations. They manifest themselves by chorea (chorea minor), or Saint Vitus' dance. Chorea is found in children and juveniles and causes involuntary convulsive contractions of the striated muscles. This disease may cause sudden death due to a convulsive constriction of the rima glottidis with resultant asphyxia.

Treatment and Prophylaxis

In the acute period, the patient should be confined to bed. The room in which he is lodged must be warm, dry and well-illuminated. The affected joints are wrapped in cottonwool. In view of frequent sweats, the clothes should be changed as often as possible and the skin wiped with cottonwool soaked in alcohol, cologne, or lukewarm water with an addition of diluted acetic acid. The joints are put in a comfortable position. For intense pains, it is recommended to apply compresses painted with analgetic ointments to the joints (formula No. 178). The best known remedy against rheumatic fever is sodium salicylate which is administered in a total daily dose of 8 to 12 g divided into 1-2 g portions given once in every 2-3 hours. The preparation may also be used in solution form (formula No. 127) and in powder form (formula No. 121). In case of poor tolerance of sodium salicylate it is replaced by aspirin in a half-dose (formula No. 122). Usually, on the fifth or sixth day of therapy there is considerable amelioration in the patient's general condition with abatement of fever and pains in the joints and better subjective feeling. The next step in treatment is administration of salicylates in half-doses with a gradual reduction by 1 g every three days. When the dose is reduced to 2 g sodium salicylate therapy is continued for another 15-20 days. In view of the fact that sodium salicylate causes heavy sweats and a sharp decline in the arterial pressure, it is recommended to administer caffeine in a dose of 0.2-0.3 g per 24 hours and injections of 20 per cent camphor once or twice daily and cordiamine in a dose of 1-2 ml once or twice daily. Other effective drugs used to reduce the body reactivity in rheumatic fever, in addition to sodium salicylate, are aspirin (4-6 g daily particularly in intolerance of sodium salicylate), aminopyrine (2-3 g per 24 hours), butadione in doses of 0.15 g three times daily, and pyrazolidine. These medicines also quickly eliminate symptoms of exudation in rheumatic fever. Aminopyrine as well as butadione are applied with a watch over the white cell count, since they are likely to cause leucopenia and agranulocytosis. Of late, rheumatism has been treated also with corticosteroid hormones—cortisone and its derivatives (cortisone in doses of 40 mg daily and its derivaties—prednisone, prednisolone in doses of 5-10 mg three times daily during a period of 2-3 weeks). ACTH is also prescribed. Hormone therapy improves water and salt metabolism, retains water and common salt, acts on connective tissue colloids, decreases the level of hyaluronidase, and contributes to an early resorption of exudate. During hormone therapy potassium chloride is administered in a daily dose of 3 g. Salicylate therapy may be combined with administration of corticosteroid hormones, ACTH or penicillin in 250,000 unit doses given 3-4 times daily.

Of the physiotherapeutic measures taken in the acute period it is recommended to administer dry air baths and ultraviolet light in erythemic doses. Paraffin, diathermy, ozocerite, light massage, and therapeutic exercises are prescribed for residual symptoms after recovery, for restoration of articular function. Rheumatic fever is prevented by improving working and living conditions through elimination of harmful factors contributing to its prevalence (infections, chilling, damp surroundings, etc.). General advancement of public welfare in the Soviet Union, availability of excellent facilities for physical culture and sports and free medical aid have provided favourable conditions for an effective control of rheumatic fever. For prophylactic purposes, a person affected by rheumatism should be helped to improve his general health, in particular the condition of the oral cavity and nasopharynx by excising the tonsils, extracting carious teeth, etc. An important role in rheumatic fever prophylaxis is played by cardiorheumatologic departments at polyclinics, where rheumatic patients are kept under regular observation and given periodical bicillin therapy.

Personal hygiene is also highly important for prophylaxis of rheumatic fever and its recurrent attacks. For this purpose, one should be careful to avoid infectious diseases, chilling, working in damp rooms, and should be dressed according to season. There is need to be always on one's guard against tonsillitis and catarrhal states. Special measures should be taken to fortify the nervous system. Mental and physical overexertion, insomnia, and other factors weakening the body may provoke a relapse of rheumatic fever.

The diet must be sufficient and rich in proteins, carbohydrates and vitamins. Common salt intake is restricted to 3-4 g per day, since it is likely to contribute to aggravation of the inflammatory process. Liquid intake should not be limited in view of intense perspiration.

Patients recovered from rheumatic fever are sent for treatment to a sanatorium or climatic resort, but not earlier than 8-9 months after an attack.

ENDOCARDITIS

Endocarditis is an inflammation of the internal (lining) membrane of the heart. Depending on its aetiology, it occurs in rheumatic and septic forms, as has been noted in clinical practice.

Rheumatic Endocarditis (endocarditis rheumatica)

Rheumatic endocarditis may be primary (endocarditis rheumatica recens) and relapsing (endocarditis rheumatica recurrens).

Pathologic anatomy. Aschoff-Talalayev's granulomas form in the valvular stroma and are subject to subsequent sclerosis. Fibrin deposits on the valve surface become adherent to the valves, especially along the line of closure of their leaflets, where thickenings and warts (veruccae) are formed. Therefore, rheumatic endocarditis is also called verrucous (warty). The sclerosed granulomas and warts cause deformities in the valves which become thickened and shorter, features interfering with their complete closure. This defect is known as valvular incompetence or insufficiency. In the presence of adhesions along the leaflet margins, the valves are unable to open completely, so there is a narrowing (stenosis). The most frequent site of affection is the mitral (bicuspid) valve, then follows the aortic valve, and, rarely, the tricuspid valve. Rheumatic fever may produce combined lesions of the valves, most commonly of the mitral and the aortic.

Clinical picture. In the majority of cases, endocarditis is preceded by tonsillitis or chilling. In respect of its clinical course, endocarditis occurs in two varieties; one of them is accompanied by polyarthritis, the other runs its course without inflammatory changes in the joints (cardiac form). The patient complains of debility, early fatigue on exertion, cardiac discomfort, and palpitation. At the same time, there is an increase in body temperature to a subfebrile level. The pulse becomes unsteady and quickened on physical exertion. There is moderate leucocytosis, an elevated ESR, while the electrocardiogram shows an impairment (deceleration) of atrioventricular conduction. The size of the heart as evidenced by percussion and x-rays remains unchanged or is slightly expanded if primary affection is the case. At the heart apex there is a soft systolic murmur which is not audible at a distance from its site. All of the above symptoms may only point to the presence of rheumatic endocarditis. Therefore, in all such cases it is better to consider rheumatic carditis without strictly defining the localization of the process. Rheumatic carditis is diagnosed with confidence only when the above-mentioned symptoms are joined by diastolic murmur heard at the apex and base of the heart and encountered with organic changes in the mitral, aortic and tricuspid valves. Murmurs arising in rheumatic carditis are characteristically variable in respect of intensity, timbre and duration, which is an important sign of an inflammatory process in the valves.

Identification of rheumatic endocarditis is very difficult, particularly in those cases where a history of rheumatic attacks and articular lesions is absent. An accurate diagnosis is possible only after complete development of the granuloma, formation of scars in the valves, with valvular incompetence and stenosis of the orifices.

Most commonly endocarditis has to be differentiated from influenza, malaria, and tuberculosis.

One should be aware of the risk of an erroneous diagnosis of endocarditis based on such facts as subfebrile temperature, systolic murmur, and complaints of uncertain character. It should be borne in mind that prolonged subfebrile temperature may result from a disturbance in thermoregulation—thermoneuroses, thyrotoxicosis, and chronic tonsillitis. In these conditions, fever is unamenabe to pyramidon therapy; there is no leucocytosis nor elevated ESR. Rheumatic endocarditis most commonly leaves a heart defect.

Endocarditis is called relapsing when valves already affected with primary rheumatic endocarditis are involved in the inflammatory process during a recurrent rheumatic attack. The patient's complaints are the same as those in primary endocarditis; objective examination, however, reveals signs of circulatory disorder, with extrasystole, auricular fibrillation, and disturbance in the conducting system of the heart. This is accounted for by the fact that in relapsing endocarditis a valvular defect and precursory changes in the heart muscle are already present. Relapsing endocarditis causes further deformation of the already affected valves or damages other valves.

Relapsing endocarditis runs its course both with and without inflammatory processes in the joints, but always with a high-grade fever. In the presence of a valvular disease, however, not any rise in body temperature may be regarded as a sign of relapsing endocarditis. In patients with a valvular lesion, an increase in temperature is often one of the signs of pulmonary infarction, thrombophlebitis, and pneumonia, encountered in circulatory disorders. A disease process running a torpid course gives no elevated ESR or fever. In recent time, new evidence to help recognize an active rheumatic process has been found—certain immunologic reactions of the body, in particular high titres of O-antistreptolysin, antihyaluronidase, etc.

Prognosis. Endocarditis is dangerous for life in children and adolescents. In adult persons it runs a favourable course, leaving one or another heart defect after recovery.

Prophylaxis and treatment. Prophylactic measures against this disease are identical with those for rheumatic fever. Personal prophylaxis consists in measures to eliminate focal infections in the tonsils, the teeth, the paranasal sinuses, the nasal cavity, etc. In the acute period, the patient is confined to bed. The body is desensitized with salicylates, pyramidon, butadione, calcium chloride, cortisone, ACTH, vitamin P, and rutin. Adonis preparations are used for cardiovascular insufficiency. The tranquillizers are bromides and tincture of valerian. In all cases, it is necessary to administer antibiotics so as to eliminate an overt or covert focus of infection. The diet should be sufficiently nourishing and rich in vitamins. Salt and liquid intake is restricted.

Bacterial Endocarditis

Acute Bacterial Endocarditis (endocarditis septica acuta)

Acute bacterial endocarditis develops as a complication of a certain primary focus of infection whose causal agents may be streptococci, staphylococci, meningococci, gonococci, *E. coli* and other organisms. A primary focus is sometimes readily diagnosed in case of its external localization, for example, in paronychia, carbuncles, or wounds, as well as after labour and miscarriage. Cases are fairly frequent, however, of a deep-seated primary focus going unrecognized. From the local focus pathogenic organisms periodically enter the general blood stream and form secondary foci of infection, including those in the endocardium. In particular, they cause a predominantly ulcerative affection of the aortic, mitral and, less often, tricuspid valves (in gonorrhoea and pneumonia).

Clinical picture. The patient complains of general debility and malaise. Fever is usually remittent, and is accompanied by a chill and profuse perspiration. The heart is enlarged, with tachycardia, arrhythmia, and hypotonia. The spleen is enlarged, soft and palpable with difficulty. Involvement of the heart is recognized only from the presence of cicatricial processes occurring in the valves due to the influence of antibiotic therapy, when systolic murmur is joined by diastolic murmur. The disease often runs its course with purulent pericarditis and pleuritis. As the disease progresses anaemia comes on the stage. The diagnosis of bacterial endocarditis is established with certainty also when the loose thrombotic masses deposited on the valves become a source of emboli carried into various organs—the spleen, the kidneys, the lungs, and the brain, giving rise to infarction or dysfunction of organs.

Treatment. Over the past few years, the principal septic process has been effectively cured with antibiotics in combination with sulpha drugs. If the principal focus is accessible to local interference, comprehensive treatment is undertaken, including surgery. Other remedies are blood transfusion, measures to improve the general health, and adequate diet.

Subacute Bacterial Endocarditis

Subacute bacterial endocarditis (endocarditis septica lenta) was for the first time described by A. Langovoi (1884) and T. Lukin (1903). It is caused by the *Streptococcus viridans* in persons hypersensitive to this organism. This hypersensitivity is produced by precursory diseases, such as rheumatic fever, syphilis leaving a valvular lesion, sclerotic and congential heart defects (patent Botallo's duct) and in arterio-venous aneurysms of both traumatic and congenital character. In some persons, subacute bacterial endocarditis may also develop as a primary lesion on intact valves. This form has been termed primary septic endocarditis by B. Chernogubov. It is most probable that subacute bacterial endocarditis is a septic disease causing further affection of the valves in the presence of old heart defects of any aetiology. Predominance of long-standing rheumatic defects in the heart of patients is due to the fact that among all known causes of heart defects the share of rheumatic fever is the largest.

Pathologic anatomy. On the valves there are fibrin deposits under which tissue becomes necrotic, disintegrates and forms ulcers (endocarditis ulcerosa). Bacterial masses accumulate in the affected tissue. In addition to ulcerations polypous masses form on the valves (polypous-ulcerative endocarditis). The endothelium of the arteries and veins is also involved in the disease process (vasculitis). Disseminated vasculitis sometimes leads to obstruction of the terminal arteries in various organs with the development of infarction and areas of dead tissue. In disintegration of ulcers particles of tissue or fibrin are carried as emboli with the blood stream to various organs in which they cause infarctions and inflammatory changes. No purulent softening is noted in such cases.

Clinical picture. The disease runs an insidious course imperceptibly for the sufferer and very slowly until the appearance of manifest clinical symptoms. The patient complains of growing debility, malaise and fever followed by exhausting perspiration. The principal sign of the disease is prolonged undulant fever, which rises in relapses and falls down to normal in remissions. Often, fever climbs up to 39-40°C after a chill and is followed by profuse perspiration.

The skin is pale and has the so-called cafe-au-lait tan. The tips of the fingers and toes are clubbed, with curvature of the nails over the finger ends and toes. These symptoms are the result of neurodystrophic changes. Sometimes the disease process is noted to give rise to a fresh heart defect, mostly aortic, less frequently, mitral and tricuspid. Affection of the vascular system—vasculitis and fragility of minute vessels—is recognized from a positive endothelial symptom (Konchalovsky's tourniquet symptom—the appearance of petechiae on the forearm below the tourniquet compressing the veins. Petechiae also erupt in the conjunctival sac, mainly on the mucous lining of the palpebral fold of the lower eyelid (Lukin's symptom). In addition, affection of the endothelium causes multiple thrombi (blood clots) to form in the kidneys, the spleen, the skin, the lungs, and other organs; haemorrhages in the skin and acute disturbances of the cerebral circulation, and inflammation of the kidneys, more often diffuse, but sometimes focal in character (haematuria) occur also. Enlargement of the spleen is one of the permanent signs. In case of infarction in the spleen, the latter enlarges to a considerable size and becomes markedly tender on palpation; simultaneously, a peritoneal friction sound (perisplenitis) may be audible over this place. Thromboses in the kidneys are attended with attacks of severe lumbar pains and excretion of blood-stained urine. In focal nephritis the renal function is not disturbed, and the arterial pressure is not heightened. The blood picture is one of considerable anaemia, leucopenia, less often moderate leucocytosis, sometimes thrombocytopenia. Among the leucocytes many monocytes and histiocytes are found, which is a sign of irritation of the reticuloendothelial system.

The blood serum shows the prevalence of globulins; therefore, upon addition of formalin (formaldehyde) the serum turns into a jelly-like mass (positive formalin test). Blood cultures during a high-grade fever may yield *Streptococcus viridans.*

The disease runs an undulating course with periodic exacerbations alternating with remissions and tends to progress slowly.

In respect of the prevalence of individual symptoms or their combinations, subacute bacterial endocarditis may run its course in several variants: (a) septic, when the major symptom is fever; (b) anaemic, with severe anaemia; (c) renal, with symptoms of renal insufficiency and uraemia; (d) cardiac, with a circulatory disturbance. The latter variant is the result of antibiotic therapy when the septic process has been brought under control at the expense of cicatricial changes in the valves, which later cause a circulatory disorder.

This disease should be differentiated from rheumatic endocarditis, syphilitic aortitis, malaria, sometimes relapsing endocarditis. Symptoms in favour of rheumatic carditis are moderate fever, a circulatory disturbance, a history of tonsillitis and polyarthritis, a negative formalin test, and absence of vasculitis with thromboses and embolism with infarctions. The difference between subacute bacterial endocarditis and syphilitic aortitis, also running its course with aortic valve insufficiency, skin pallor, fever, and cerebral symptoms, consists in that the former is characterized by considerable anaemia, thromboses, and a positive formalin test. Wassermann's test is not of decisive importance since it may be positive also in subacute bacterial endocarditis. In differentiation from malaria, which involves fever, chills and profuse perspiration, anaemia and leucopenia, monocytosis, and splenomegaly, one should bear in mind that at the same time the blood is found to contain plasmodia, the cardiac valves are unchanged, and the symptom of clubbed fingers and toes and curvature over the nails is absent.

At present, much evidence is available on the possibility of curing subacute bacterial endocarditis by an early administration of massive doses of antibiotics.

Death may result from arterial thrombosis or embolism of vitally important organs, azotaemic uraemia and a circulatory disturbance.

Treatment and prophylaxis. Control of rheumatism fever and infection, protection of patients with heart defects against infection by means of preventive penicillin therapy are the basis for prophylaxis.

Patients should be strictly confined to bed irrespective of their subjective symptoms. Penicillin is administered intramuscularly in a dose of 800,000-1,000,000 units per 24 hours in combination with streptomycin or levomycetin during a period of 6-8 weeks followed by another treatment course after a brief interval. In antibiotic-resistant forms of subacute bacterial endocarditis it is recommended to use large doses (100 g and more for a treatment course) of sulfonamide preparations. Biomycin is also indicated in a dose of 100,000 units given 3-4 times daily during a period of 4-5 weeks. Simultaneously, measures to improve the general health and desensitizing therapy are applied by administration of salicylates (aspirin), pyramidon, and calcium chloride. In affections of the kidneys, a diet with a restricted intake of extractive substances is prescribed. For a circulatory disorder, cardiac drugs are administered, while salt and liquid intake is limited.

DEFECTS OF THE CARDIAC VALVES (VITIUM CORDIS)

Heart defects result from anatomic dislocations in the heart valves caused by rheumatic, sclerotic and syphilitic processes which disturb the valve function and lead to a circulatory disturbance at a certain stage of the disease process.

These defects may be congenital, acquired, and traumatic in origin. Congenital defects, just as traumatic ones, are encountered very rarely. The former develop during intrauterine life as a result of abnormal growth and formation of the cardiovascular system. They also occur as sequels to inflammatory processes in the right heart sustained during intrauterine life. Among congenital defects are patency of Botallo's duct which during intrauterine life connects the pulmonary artery with the aorta, patency of the oval opening which is present in the interatrial septum of the foetus and which normally closes after birth, a defect in the interventricular septum, stenosis of the mouth of the pulmonary artery, etc. Defects of the cardiac valves are for the most part acquired.

Mitral Valve Insufficiency (insufficientia v. mitralis)

etiology and pathogenesis. Insufficiency of the mitral valve develops as a result of anatomic changes in the valve as well as in the tendinous fibres attached to it, which causes imperfect closure of the valves guarding the left atrioventricular orifice. In insufficiency of the mitral valve, during a systole the blood is delivered not only into the aorta but also into the left atrium into which the blood from the pulmonary artery is delivered simultaneously. This results in a greater than normal amount of blood in the left atrium, which

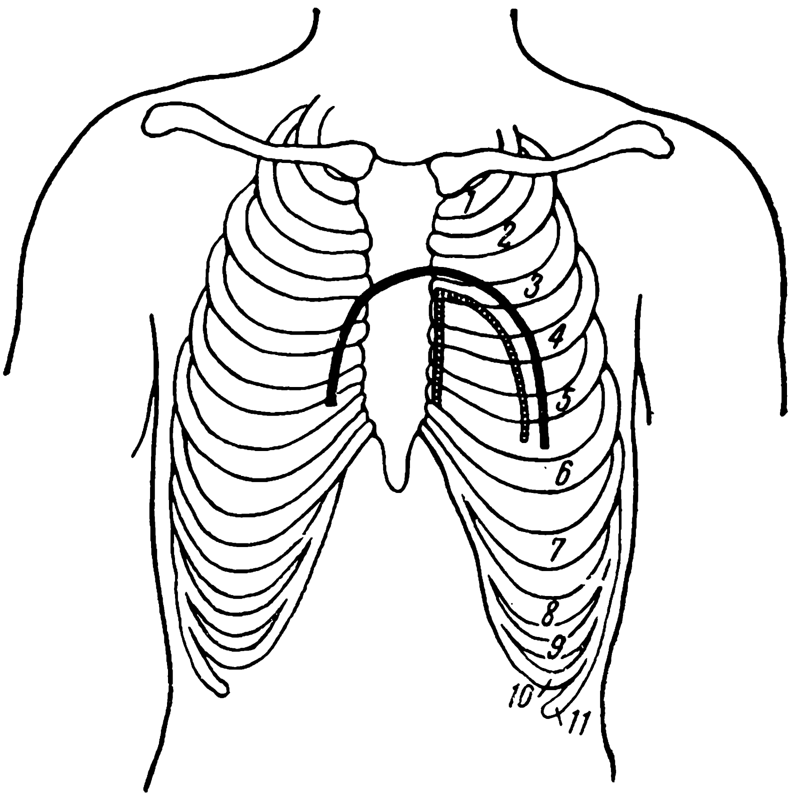

Fig. 39. Heart borders in mitral defect

leads to its distention and hypertrophy of its muscle. During a diastole the left ventricle, instead of receiving 50-60 ml of blood, as is normally the case, receives also that amount which entered the left atrium because of mitral valve insufficiency. This in its turn leads to distention of the left ventricle with a later hypertrophy. Disturbed blood circulation is brought to normal by an increased work of the hypertrophied left ventricle. In time, however, the muscle of the left ventricle weakens, the blood drainage from the atrium is made difficult, the pressure within it is increased due to congestion of the blood, which leads to stasis in the pulmonary circulation. The latter in its turn increases the work of the right ventricle which becomes hypertrophied and then distended. Stasis soon extends through the veins of the general circulation.

Clinical picture. The patient at first complains of slight dyspnoea only on physical overexertion. Later, with a considerable circulatory disturbance, there are complaints of dyspnoea at rest, pains in the right hypochondrium, oedema, and enlargement of the abdomen. The apex beat is intensified due to hypertrophy of the left ventricle. Cardiac dullness is extended to the left owing to hypertrophy and distention of the left ventricle and upwards due to enlargement of the left atrium. In far-advanced cases, there is an enlargement of the heart also to the right owing to hypertrophy and distention of the right ventricle (Fig. 39). The first sound at the heart apex is reduced as a result of the absence of the closed-valve period as well as excessive blood volume in the left ventricle. It is known that in quick contraction the first sound is intensified and in slow contraction, reduced. At the heart apex a harsh systolic murmur is heard, which sometimes may be musical, and is transmitted to the left armpit and the back. This murmur is caused by the blood being partly forced back during a systole from the left ventricle into the left atrium through imperfectly closed valves. The second sound on the pulmonary artery is accentuated in consequence of pulmonary congestion and a rise in the pressure which closes the valves of the pulmonary artery.

Differential diagnosis should be made between organic systolic and functional murmurs, since a systolic murmur at the heart apex may be heard also in the absence of a defect. Organic insufficiency of the mitral valve is characterized by a coarse and musical systolic murmur, which is constant, intensified on physical exertion and equally audible when the patient is standing and lying down. In addition, the murmur is better audible at the heart apex or over the site of attachment of the 4th left costal cartilage to the sternum, and is transmitted to the left axillary region. Functional systolic murmurs are characteristically blowing and inconstant, and may disappear when the patient is standing; they weaken on physical exertion and are transmitted poorly.

In the course of bicuspid valve insufficiency there is a stage of compensation at the expense of the left ventricle and atrium, a stage of insufficiency of the left atrium with pulmonary congestion and hypertrophy of the right ventricle and, finally, a stage of insufficiency of the right ventricle with stasis in the veins of the general circulation and in the organs of the abdominal cavity. The patient's condition and findings of objective examination vary in accordance with the above-mentioned stages.

Prognosis. Prognosis depends on the state of the heart muscle. Infection and physical exertion may lead to decompensation. The gravity of the course is determined, apart from subjective symptoms, from lessening of the first sound at the heart apex and a considerable distention of the left ventricle and hypertrophy of the right ventricle. Progressive reduction in the strength of the heart muscle makes prognosis unfavourable.

Stenosis of the Left Atrioventricular Orifice (stenosis ostii venosi sinistri)

Aetiology and pathogenesis. This stenosis occurs in combination with mitral valve insufficiency as a result of rheumatic endocarditis which causes constriction of the orifice due to cicatrization of granulomas and fusion of the valve leaflets along their margins. These changes interfere with the blood flow from the left atrium into the left ventricle during a diastole and the atrium fails to expel all the blood it contains. The blood retained in the atrium is supplemented with blood supplied from the pulmonary veins. This causes distention of the left atrium. Normal circulation of the blood is maintained for some time by intensified contractions of the atrium, but the atrial muscle is soon weakened, which entails pulmonary congestion leading in its turn to hypertrophy and then dilatation of the right ventricle with stasis in the veins and organs of the general circulation. At the same time, the left ventricle undergoes no change, since it receives a small quantity of blood because of stenosis of the orifice.

Clinical picture. The patient complains of severe breathlessness and palpitation on the slightest physical exertion, precordial pains and periodic blood-spitting. On inspection marked cyanosis of the tip of the nose, lips, ear lobes, and feet (acrocyanosis) is noted.

The apex beat is unchanged, marked pulsation is notable in the epigastrium due to hypertrophy and dilatation of the right ventricle. On palpation of the precordium vibration of the thoracic wall is felt—the so-called purring thrill. The area of cardiac dullness is extended upwards and to the right. The first sound at the heart apex is flapping and loud, apparently because of a blood deficit in the left ventricle. The accentuated second sound over the pulmonary artery which is audible in this defect is due to the clap made by the sharp closure of the valves of the pulmonary artery, caused by an increased pressure in the pulmonary circulation. The second sound on the pulmonary artery may be split ("quail rhythm") because of asynchronous closure of the aortic and pulmonary valves due to a heightened pressure in the pulmonary artery. At the heart apex a diastolic murmur is audible which may be louder at the beginning of a diastole and then is reduced to produce the greatest sonority at the end of the diastole, i.e., before a systole. That is why this murmur is termed presystolic. The presystolic murmur is short and rumbling and ends in a flapping first sound. The variation in murmur intensity in different phases of a diastole is accounted for by a change in the speed of the blood flow. At the beginning of a diastole the blood current is accelerated because of the great difference in pressure between the atrium and the ventricle. In the middle of the diastole, when the pressure between the atrium and the ventricle is equalized, and a little blood is retained in the atrium, the blood flow is decelerated and the murmur disappears. At the end of the diastole, however, the blood flow from the atrium into the ventricle is again accelerated because of the onset of atrial systole increasing the speed of blood expulsion, which is responsible for the intensified murmur. In severe stenosis the murmur may be absent altogether.

In stenosis of the atrioventricular orifice the pulse is of a low volume and sometimes arrhythmic. At the same time, atrial fibrillation is sometimes notable, which is caused by overdistention of the left atrium with venous blood. In this case no diastolic murmur is heard.

Stenosis of the mitral orifice is the most severe defect in respect of prognosis, since the weak muscles of the left atrium and the right ventricle which compensate for the defect are unable to cope with the disturbed circulation of the blood. It should be added that the onset of decompensation in this case is accounted for not only by the mechanical overloading of the heart but also by a concomitant rheumatic heart lesion.

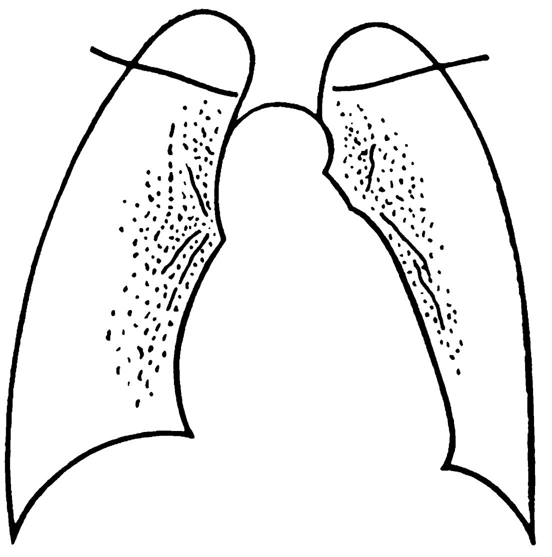
Fig. 40. X-ray of mitral stenosis

The x-ray shows a mitral configuration of the heart, i.e., the angle between the vascular bundle and the left ventricle, the left atrium and the arch of the pulmonary artery is smoothed out. The left ventricle is not enlarged; there is an expansion of the heart contours to the right due to enlargement of the right ventricle and the right atrium (Fig. 40).

In the early stage, the defect runs an insidious course, causing no unpleasant sensations, and is sometimes an accidental finding at x-ray examination, when an enlargement of the left atrium is discovered; the stage of left atrial insufficiency with stasis of the blood in the lungs is characterized by profuse blood-spitting on physical exertion. In the stage of right ventricular insufficiency due to stasis of the blood in the organs of the general circulation pains appear in the liver region and oedema as well as ascitis; dyspnoea and blood-spitting are unmarked or absent altogether. In this phase, relative tricuspid valve insufficiency comes on the stage—the accentuation and splitting of the second sound disappear since with the weakened right heart and a smaller amount of blood delivered into the pulmonary circulation the stasis within the latter comes to an end. The intensity of the diastolic murmur and purring thrill is reduced due to a slow-down in the speed of the blood flow from the left atrium into the left ventricle.

Among the complications attention is deserved by embolism in the brain with a lethal outcome or with the onset of paralysis, embolism in the retinal artery with a temporary or permanent blindness, as well as embolism in the kidneys, the spleen and the extremities, with the development of gangrene. Embolism may occur even in the early stages of a defect as a result of fragments being torn off from the thrombus in the left auricular appendage, later from the mural and spherical thrombi in the dilated left atrium. The thrombi are torn off more readily after administrations of digitalis, strophanthin, which fact should be kept in mind when prescribing a treatment, particularly in cardiac fibrillation which is above all responsible for the formation of these mural thrombi. The latter, when torn off from the wall, may freely rotate in the left atrium, become ball-shaped and obstruct the left atrioventricular orifice, which causes an exceedingly severe cyanosis and sometimes immediate death.

Haemoptysis is highly characteristic of mitral stenosis in the stage of pulmonary congestion and may occur in the form of blood streaks and blood-stained sputum and sometimes as a profuse haemorrhage caused by a rupture of vessels in the pulmonary circulation. The blood in the alveoli is absorbed by histiocytes which are discharged with sputum as heart-disease cells. Sometimes, haemoptysis is observed also in pulmonary infarction arising from occlusion of pulmonary vessels by fragments of a thrombus brought from the right atrium and the deep-lying femoral veins and by local thrombi formed in the pulmonary vessels as a result of a decelerated blood flow.

Patients with mitral stenosis are liable to attacks of cardiac asthma on physical overexertion in the period of compensatory hypertonia of the right ventricle. Cardiac asthma develops as a result of intensified delivery of blood into the pulmonary circulation, its repletion and pressure increase within it, with defective drainage through the constricted left atrioventricular orifice.

In the final phase of stenosis of the left atrioventricular orifice the liver and the lungs undergo cirrhotic changes secondary to a prolonged stasis of blood within them.

In the same stage, the skin shows brown pigmentation and trophic ulcers. The above-mentioned changes are largely due to a neurotrophic disturbance which causes a derangement in the nutrition of organs. A reduction in the protein content of the blood in its turn aggravates oedema of tissues.

An increased body temperature, even as slight as 37.5°C, deserves keen attention since it may be a sign of endocarditis, infarction, marantic thrombophlebitis, erysipelas, abscesses following injections of camphor, etc. In this condition the ESR is decelerated as a result of congestion. The appearance of jaundice may point to pulmonary infarction or pathologic changes in the congested liver. Dyspeptic symptoms arise from stasis in the gastrointestinal tract.

Insufficiency of the Aortic Valves (insufficientia valvularum aortae)

Aetiology and pathogenesis. Aortic valve insufficiency is a condition secondary to rheumatic fever, syphilis, subacute bacterial endocarditis, and extension of a sclerotic process from the aorta to the valves. At a young age, insufficiency of the aortic valves is, as a rule, of rheumatic origin. At an advanced age the first thought should be given to syphilis or aortic sclerosis. Rheumatic insufficiency of the aortic valves combines with stenosis of the aortic orifice or with a mitral defect, whereas insufficiency of syphilitic origin is most commonly isolated. In consequence of the changes produced by the above-mentioned processes, the valves are unable to close the orifice completely during the diastole, so there is a certain backflow of blood from the aorta into the left ventricle into which blood is simultaneously supplied from the left atrium also. Since it receives blood in excess, the left ventricle has to perform greater work than normally and becomes hypertrophied.

Pathologic anatomy. In rheumatic fever the aortic valves undergo considerable changes. They become shorter, with adhesions along the margins. In syphilis, the middle coat of the aorta is affected, the disease process involving the initial portion of the aorta, which becomes dilated and the valves relatively defective.

Clinical picture. The patient complains of dizziness, headaches, palpitation, precordial pains and sensations of pulsation in the neck and head. These symptoms are finally joined by dyspnoea. On inspection, pallor of the skin is notable, which, however, is not a sign of anaemia but is due to a blood deficit in the arterial vessels of the skin in the period of a cardiac diastole. There is dance of the carotids (pulsation of the carotid arteries). The apex beat is intensified and is visible to the eye. It is displaced to the left and downwards and is found in the sixth or seventh intercostal space, outwardly to the midclavicular line. During each systole, the apex of the left ventricle forcibly elevates the finger or stethoscope applied to it.

On percussion there is an increase in the area of cardiac dullness to the left (Fig. 41.) On auscultation the first sound at the apex is lessened because of the absence of the period of closed valves, the second sound over the aorta is weakened due to imperfect closure of the aortic valves. At the heart base, in the second intercostal space to the right of the sternum, or in Botkin's point, a diastolic murmur is audible which is due to a backflow of blood from the aorta into the left ventricle. The murmur is louder at the beginning of a diastole and softer at the end. Sometimes, a systolic and a diastolic murmur are heard over the heart apex. The systolic murmur is accounted for by the relative insufficiency of the mitral valve caused by distention of the left fibrous ring with a considerable hypertrophy of the left ventricle; the appearance of diastolic or rather presystolic murmur at the heart apex is explained in different ways. Apparently, a strong flow of blood from the aorta into the ventricle raises the mitral valve leaflet lying close to the aorta thereby causing stenosis of the left atrioventricular orifice.

In insufficiency of the aortic valves, one can note sharp pulsatory vibrations of both small and large vessels. As a result of these pulsations, a large full and bounding pulse is felt over the radial artery (pulsus celer et altus). The systolic arterial pressure is heightened, while the diastolic pressure is reduced, the degree of reduction

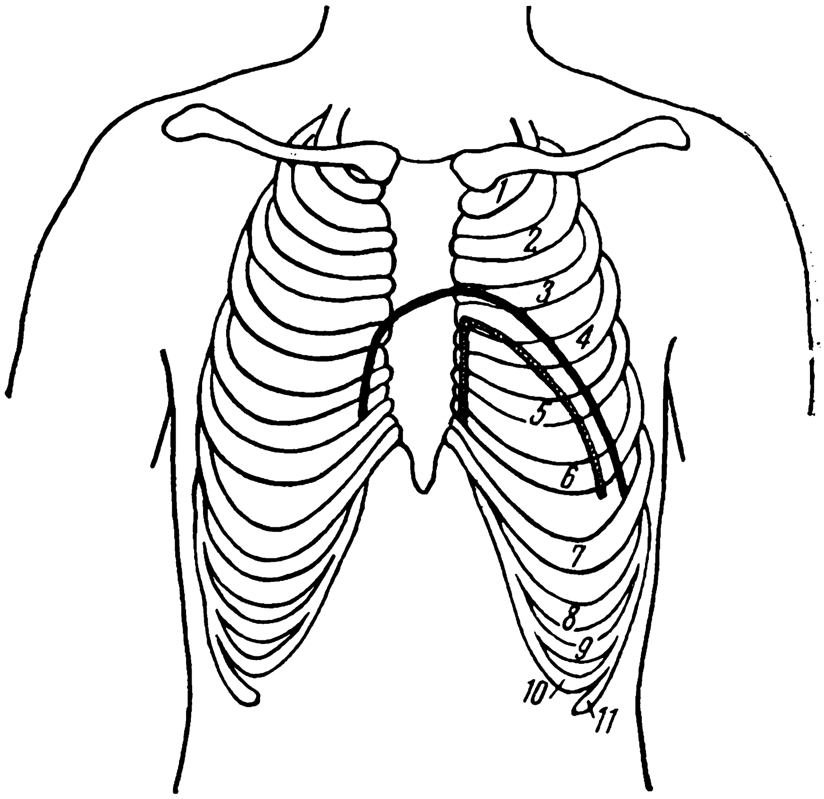

Fig. 41. Borders of the heart in aortic defect

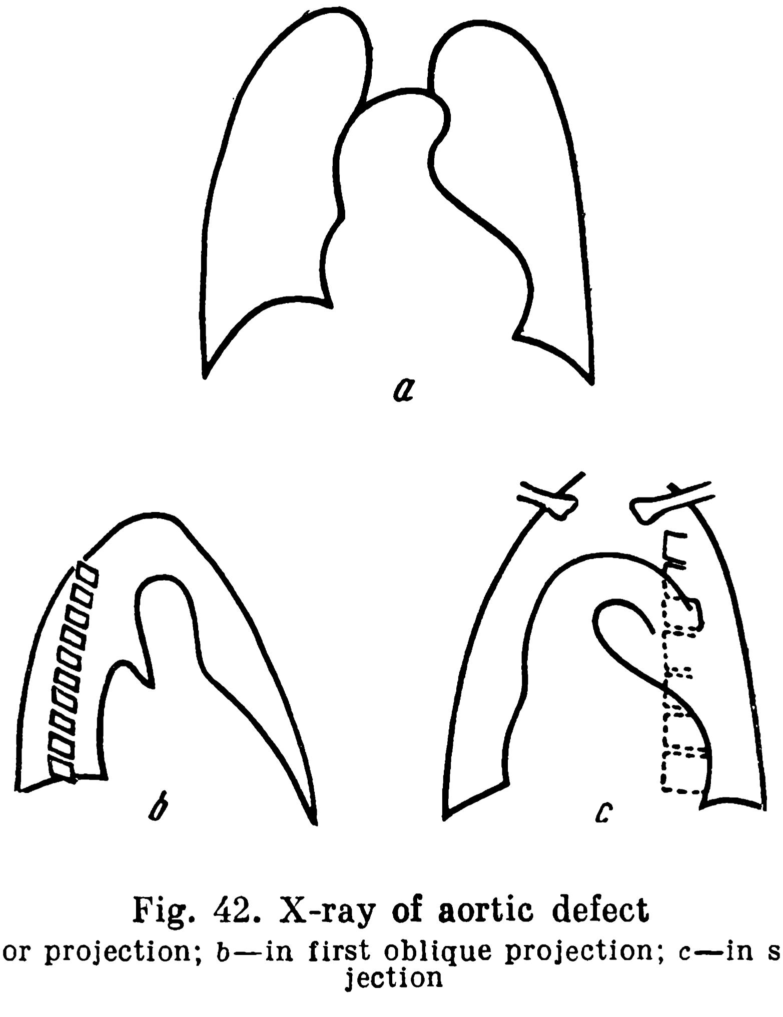

Fig. 42. X-ray of aortic defect

a—in anteroposterior projection; *b*—in first oblique projection; *c*—in second oblique projection being in direct relation to the degree of incompetence of the aortic valves.

Other characteristic signs are rhythmic pulsatory movements of the head called Musset's sign after a French poet who suffered from aortic valve insufficiency, as well as a pulsatory jerk of one leg when thrown over the other, and rhythmic dilatation and narrowing of the pupils, caused by a strong inflow and outflow of blood in the retina. In aortic valve insufficiency, sounds and murmurs are audible over large peripheral arteries. Traube's double sound is heard upon the application of a stethoscope to the femoral artery; a double Duroziez's murmur is also audible upon pressure, which corresponds to ventricular systole and diastole. The appearance of sounds is explained by a diminished tone over the arterial wall in incomplete diastolic filling of the arteries, and the double murmur, by the double flow of blood through the vessel narrowed due to compression. During a systole the blood flows toward the periphery, and during a diastole, partially back into the left ventricle. The same cause is responsible for the capillary pulse which is encountered in insufficiency of the aortic valves.

The x-ray displays a considerable enlargement of the left ventricle with a rounded heart apex and a distinctly outlined angle between the vascular bundle and the contour of the heart shadow—the aortic configuration (Fig. 42).

In aortic insufficiency, a circulatory disturbance occurs much later than in mitral failure, since compensation is provided by the strong left ventricle. Insufficiency of the aortic valves of syphilitic origin runs a less favourable course because of the impaired conditions of nutrition of the heart muscle.

Stenosis of the Aortic Orifice (stenosis ostii aortae)

An isolated stenosis of the aortic orifice is a very rare condition, and is more often seen in combination with rheumatic insufficiency of the aortic valves.

Clinical picture. The patient complains of cardiac pains caused by inadequate filling of the coronary vessels, and syncope, dyspnoea and palpitation. The skin shows pallor due to insufficient supply of blood to the peripheral vessels. The apex beat is elevating, displaced to the left and downwards, though to a far less extent than in aortic valve insufficiency. The area of cardiac dullness is enlarged to the left as a result of hypertrophy of the left ventricle. In the second intercostal space to the right of the sternum a rough systolic murmur is audible, which is readily transmitted by the blood stream to the vessels of the neck. The murmur is produced by the passage of blood through the narrowed orifice into the aorta during a systole. The pulse is rare, within 50-60 beats per min, and slow (pulsus rarus et tardus), rises slowly and is of a low volume (pulsus parvus), which is explained by a slow blood flow from the ventricle to the aorta. The arterial pressure is diminished. There is a systolic purring thrill to the right of the sternal margin in the second intercostal space.

Stenosis of the aortic orifice should not be confused with stenosis of the aorta itself anywhere along its course as well as with its dilatation, when the aortic orifice is relatively narrowed in comparison with the dilated aorta. Sometimes, in marked hypertrophy of the left ventricle stenosis of the aortic orifice has to be differentiated from hypertensive vascular disease, which, as distinct from stenosis of the aortic orifice, runs its course with hypertension and a considerable accentuation of the second sound over the aorta.

Insufficiency of the Tricuspid Valve (insufficientia valvulae tricuspidalis)

Aetiology and pathogenesis. This defect may be functional or organic in origin. Functional, or relative, insufficiency of the tricuspid valve is consequent to dilatation of the right ventricle and distention of the fibrous ring resulting in imperfect closure of the orifice by the valves. It is encountered in decompensated mitral defects as well as in rheumatic myocarditis involving no valvular lesions, and in various affections of the muscles of the right heart, in particular in an isolated affection of the right heart due to changes in the lungs (cor pulmonale—pulmonary heart). Much less frequently this defect is organic, being consequent to rheumatic endocarditis combined with a mitral and aortic defect. An isolated organic defect of the tricuspid valve is a very rare phenomenon. In incompetence of the tricuspid valve during a systole the blood is forced not only into the pulmonary circulation through the pulmonary artery but also into the right atrium, causing the latter to enlarge considerably.

Clinical picture. The presence of dyspnoea is a very early complaint. On inspection there is marked cyanosis with an icteric hue, and pulsation in the epigastrium due to enlargement of the right ventricle. The enlarged and hard liver also pulsates in the systolic phase (systolic pulsation of the liver). Pulsation of the veins on the neck and liver is due to the entry of blood during a systole from the right ventricle into the right atrium because of imperfect closure of the tricuspid valve, and from there into the venae cava superior and inferior. Because of stasis in the veins of the general circulation and the organs of the abdominal cavity there is considerable oedema in the lower extremities, and ascitis. On percussion, the area of cardiac dullness is enlarged to the right. At the heart apex, near the xiphoid process of the sternum, there is a systolic murmur, which is especially well audible behind the sternum, at the level of attachment of the 4th rib. This defect involves no stasis in the pulmonary circulation since a considerable part of the blood delivered from the right ventricle to the liver and back does not take part in the normal blood circulation and relieves pulmonary congestion.

In organic defects of the tricuspid valve the positive venous pulse and systolic pulsation of the liver fail to disappear after the onset of compensation, while with a relative functional insufficiency all these symptoms cease to exist upon compensation.

Combined Heart Defects

Isolated defects of individual valves are less frequent than combined defects. In clinical practice insufficiency of the mitral valve is often concurrent with stenosis of the left atrioventricular orifice, insufficiency of the aortic valves of rheumatic origin combines with stenosis of the aortic orifice, etc. This is explained by the rheumatic process, in addition to valvular deformity, also causing adhesions of the valve margins, with resulting stenosis of the orifice. Valve insufficiency occurs ahead of stenosis in time. In combined defects symptoms of insufficiency prevail in the majority of cases in the first period of the disease, and symptoms of stenosis come to the fore later. In addition to a combined defect of one cardiac valve, for example, the mitral valve, cases of insufficiency of two and three valves are fairly frequent. For example, a defect of the bicuspid valve occurs in combination with those of the tricuspid and aortic valves. In combined defects symptoms of one of the defects predominate in the clinical picture. The practical import of these combined defects of one, two and three valves is substantial, since the picture of the disease in such cases is subject to a certain change.

In combined defects there is sometimes a certain compensation of circulatory disorder. For example, in extensive circulatory disturbance caused by a defective bicuspid valve, the supervention of tricuspid valve insufficiency brings relief since part of the blood is drained into the veins of the general circulation, pulmonary stasis is considerably reduced, and dyspnoea is diminished while less blood is supplied into the left ventricle, which, therefore, suffers no overload, with a resulting temporary delay in its dilation. The co-existence of mitral valve insufficiency and stenosis of the left atrioventricular orifice aggravates the course of the disease and leads to an earlier decompensation.

Heart Disease and Pregnancy

A woman with a heart disease is sometimes advised to avoid pregnancy with its increased requirements in respect of the heart which the latter is not always capable of satisfying. This increased load on the heart results from a growth of the vascular network in the enlarged uterus: in addition the growing foetus demands a considerable amount of additional oxygen while the high position of the diaphragm in connection with pregnancy impairs the conditions of the heart's action. In the case of pregnancy in a woman with a heart defect one has to consider the possibility of labour or the need for termination of pregnancy. The absolute indications for abortion are as follows: the presence of active rheumatic endocarditis, defects of any cardiac valve, decompensated or with a past history of decompensation, diseases of the myocardium and pericardium, myocardial infarctions, both fresh and healed, quickly progressing forms of hypertensive vascular disease, and cardiopulmonary insufficiency.

Not every organic defect requires termination of pregnancy, since many women with mitral valve insufficiency have labour without any difficulties or complications. Judgement on the question of artificial abortion should not be based on such findings as a systolic murmur heard at the heart apex and a change in the heart configuration, since the murmur is audible even under normal conditions at the apex of the heart in many women, and in pregnancy its appearance may be due to physiologic features of the body. Therefore, before passing judgement on a heart defect in a pregnant woman, one has to take into consideration not only physical but also laboratory and instrumental findings, as well as the case history, i.e., infectious diseases sustained in the past, particularly rheumatic fever. If appropriate indications are available, pregnancy should be interrupted not later than in 8 weeks; at late periods, the delivery is effected by surgery (Caesarean section). Women with a heart defect allowed to have a bady should be under medical observation, and their early hospitalization is desirable, approximately 4-6 weeks before labour. Treatment of pregnant women with symptoms of decompensation is carried out in accordance with the same general rules as in heart disease.

Prophylaxis and Treatment of Heart Defects

The development of heart defects is prevented by measures to control rheumatic fever, atherosclerosis and syphilis, as well as by general measures to improve the condition of the oral cavity and the nasopharynx, e.g., treatment of chronic tonsillitis, extraction of carious teeth, etc. In the presence of organic changes in the valves and their orifices one has to find out whether they are accompanied by a circulatory disturbance. Defects not involving the latter are termed compensated. Patients with compensated defects are fully ablebodied and may be considered practically healthy. In these cases, preservation of working capacity is the principal task. This task is fulfilled by the following measures: (a) protection of the patient against various infections, particularly attacks of rheumatic fever; (b) detection and elimination of all foci of infection in the nasopharynx, the oral cavity, and female genitalia, since under certain conditions they may become the source of microorganisms invading the blood stream and affecting the heart. In addition, each patient with a heart defect should be prescribed an individual routine of life conforming to the condition of his heart and his personal traits and intended to spare him any strain on his nervous system whenever possible. While enjoining the patient to avoid physical and mental overexertion which is likely to cause a disturbance in circulation of the blood, he is advised to engage in light physical work, which trains the heart and vessels and has a favourable influence on the blood circulation. Other requirements are sufficient sleep at night and sometimes in the daytime, moderation in food and liquid intake, abstinence from tobacco and liquors. Persons with a heart defect should be kept under constant medical observation so as to be able to prevent the onset of decompensation by timely hospitalization or appropriate medical treatment.

The causes of decompensation are infectious diseases as well as attacks of rheumatic fever, physical overexertion, and various unhealthful conditions of life producing a marked weakening of the heart muscle and impairment of the flow of blood. The first signs of decompensation are dyspnoea and palpitation on physical exertion, then pains and distressing sensations in the heart region, and quickened pulse. All these symptoms pass with the cessation of strain. In the subsequent development of a circulatory failure the abovementioned symptoms are joined by symptoms of stasis in the pulmonary and general circulation. For example, in stenosis of the left atrioventricular orifice there is pulmonary congestion, which is manifested by congestive bronchitis, dry and moist rales in the lungs, particularly in the lower lobes, haemoptysis, the presence of heart-disease cells in sputum, and an intensified second sound over the pulmonary artery. Subsequently, with a weakening of the right ventricle, stasis in the pulmonary circulation is lessened owing to retention of a certain part of the blood in the veins of the general circulation lying close to the heart. The veins on the neck become engorged and show pulsation, stasis develops in the liver, which is manifested by an enlargement and tenderness of the latter as a result of distention of its capsule, and by its systolic pulsation. The venous pressure in such cases is considerably increased and may reach 200 mm of water and more instead of the normal 100 mm. Dilatation of the engorged veins and capillaries causes a considerable deceleration of the blood flow, oxygen deficiency and cyanosis of the extremities—acrocyanosis. Later, oedema in the lower extremities increases and becomes permanent, invades the entire subcutaneous tissue, and later ascitis, hydrothorax, and hydropericardium come on the stage. In a circulatory disturbance, the urine discharge is markedly diminished, the urine acquires a high specific gravity, and is found to contain protein and erythrocytes. In a considerable failure of the blood circulation the patient finds relief only in the sitting or semirecumbent position (orthopnoea) on a bed, couch or armchair, with his legs hanging down; and even sleeps in this posture. In this period, the heart sounds are muffled, the heart boundaries are considerably expanded, the murmurs are weakened or cease altogether, and various types of arrhythmia occur. Nutrition of all organs is impaired and cachexia (emaciation) often develops.

In respect of its manifestations, cardiac insufficiency is known to develop in three stages. In the first stage the patient complains of breathlessness and palpitation only on physical exertion; symptoms of congestion are absent. In the second stage the heart gives cause for complaints even in performance of usual work, with stasis in the pulmonary circulation and the liver and reduced working capacity. In the third stage dyspnoea and palpitation appear even at rest, while symptoms of congestion in the pulmonary and general circulation become worse than in the second stage. Irreversible cirrhotic changes simultaneously develop in various organs, particularly in the lungs and the liver, as well as ascitis, hydrothorax, and symptoms of stasis in the gastrointestinal tract. The patient in this period is incapacitated and needs nursing care.

Upon the onset of the first signs of cardiac insufficiency—dyspnoea, palpitation during usual work, oedema in the legs in the evening and a reduction in urinary secretion — the patient is confined to bed and enjoined to rest, which is sometimes quite sufficient for restoring adequate circulation of the blood. With a more severe circulatory insufficiency, when dyspnoea, palpitation and cyanosis are more manifest, drug therapy is undertaken.

Drug therapy. Of the cardiac drugs the most effective is **digitalis (foxglove)**, which has an effect not only on the muscle of the heart but also on its conducting system as well as on the vessels. Digitalis slows down the pulse rate and the conduction of impulses from the sinus node, lengthens the diastole and intensifies the systole. Under the influence of digitalis, the renal vessels are dilated and the vessels of the abdominal cavity are markedly constricted. As a result, dyspnoea and symptoms of congestion in organs are reduced, and the amount of urinary secretion is considerably increased. Digitalis produces untoward effects also. It enhances the excitability of the heart muscle and may provoke premature heart contractions (extrasystole) of the bigeminal type, when every normal contraction is followed by an extrasystole. The appearance of bigeminy is a serious signal for withdrawing digitalis, otherwise ventricular fibrillation may arise with a later sudden death of the patient. Digitalis causes constriction of the coronary vessels. Therefore, in predisposition to coronary spasm its application is contraindicated. Digitalis is excreted from the body with difficulty, therefore, its prolonged application in large doses leads to intoxication. Digitalis is prescribed in small doses—from 0.1 to 0.15 g per 24 hours—on condition that the patient is kept under constant observation.

Digitalis is helpful in decompensated defects of the heart, and in the tachycardiac form of atrial fibrillation. Contraindications for the administration of digitalis are coronary sclerosis with attacks of stenocardia, and partial and complete heart block. It is forbidden to use digitalis in rheumatic carditis and acute myocarditis, since they affect the conducting system and hold out the danger of a heart block. Moreover, in endocarditis, under the influence of intensified contractions of the heart fragments may be torn off from the valves and fibrin deposits and carried as emboli into various organs. Digitalis may be used in powder form (formula No. 1), in suppositories (formula No. 2), and in solution form (formula No. 3). When administered per os digitalis gives an effect in 2-3 days, and upon its withdrawal its action lasts for about a week. A knowledge of these terms is very important for prevention of a cumulative effect. When administration of digitalis is followed by vomiting and nausea as well as in severe stasis in the gastrointestinal tract as a result of inadequate absorption of the drug it is recommended in suppositories or in solution form (enema) (formula No. 4). For subcutaneous or intravenous injections digalen-neo is used (formula No. 7).

Another drug used in congestive heart failure is **strophanthin**, a digitalis-like preparation. For its effect this drug differs from the latter in that its action is faster, particularly upon intravenous administration; therefore, it is especially helpful in case of emergency. It does not cause constriction of the coronary vessels, which makes it indispensable for congestive cardiac failure associated with coronary cardiosclerosis, neither does it produce intoxication since it is very quickly broken up and eliminated from the body.

The application of strophanthin may be immediately followed by administrations of digitalis, but the use of strophanthin is permitted not earlier than a week after digitalis has been discontinued when the latter has been completely eliminated from the body. Intravenously strophanthin is introduced once or twice daily (formula No. 12). In combination with 20 ml of a 40 per cent glucose solution it is injected into the vein very slowly, during 3-5 minutes. Also suitable for intravenous injection is the ordinary strophanthin tincture in a dose of 2-3 drops in a physiologic salt solution or in 20 ml of a 40 per cent glucose solution, as well as corglycon in a dose of 1 ml to 20 ml of a 40 per cent glucose solution (formula No. 13).

Adonis (Adonis vernalis) was introduced into clinical practice by N. Bubnov, a follower of S. Botkin. In action it is similar to but weaker than digitalis. It is an effective diuretic and causes no intoxication; therefore, it is suitable for a prolonged administration (formula No. 8). For improving circulation of the blood wide use is made of a combination of bromides with codeine and adonis proposed by V. Bechterew for the treatment of epilepsy (formula No. 118). For adjustment of the higher nervous system in patients with cardiac disease and for enhancement of defensive inhibition processes it is recommended to use bromides (formula No. 114) and barbiturate preparations (formulas Nos. 110, 111, 112).

Camphor is widely used for diseases of the cardiovascular system, particularly in those cases where the application of digitalis is contraindicated. Sometimes, a subcutaneous injection of camphor prepared on low-quality oils gives rise to infiltrates—camphoromas—which resist resorption for a long time, may become infected and produce phlegmons and abscesses. The ordinary oil solution of camphor is unfit for intravenous infusion since it is liable to cause a fatal embolism. Corasole (pentylene tetrazole), cordiazol (metrazol) (formula No. 38), and cordiamine (nikethamine) (formula No. 34) are widely used to strengthen the heart muscle.

Caffeine is used as a remedy stimulating cardiac and cerebral activity and dilating the cerebral, coronary and renal vessels. On a subcutaneous injection its action is instantaneous; on per os administration its absorption and action are rapid.

Strychnine. A 0.1 per cent solution of strychnine nitrate in a dose of 1 ml injected subcutaneously improves the tone of the heart muscle (formula No. 37). It may also be used in tincture form (formula No. 41).

Glucose is widely used for the treatment of heart disease by its intravenous injections in a hypertonic solution. Glucose is a perfect nutritive material for the heart muscle, improving its contractile capacity and producing a diuretic effect, taking up water from tissues and increasing the blood volume. It is administered intravenously in a dose of 20-30 ml of a 40 per cent solution during a period of 20-30 days (formula No. 42).

Oxygen therapy is especially helpful in cardiopulmonary insufficiency, when arterialization of the blood in the pulmonary circulation is insufficient because of the destruction of capillaries. Oxygen is also indicated for a considerable pulmonary congestion, and for cirrhotic changes in the lungs.

Blood-letting in case of an increase in the volume of circulating blood (in pulmonary oedema) in a quantity of 300-400 ml considerably improves the patient's general condition. Blood-letting is contraindicated in the dystrophic stage of congestive heart failure consequent to anaemia, cachexia and hypoproteinaemia.

Leeches, six to eight in number, applied to the region of the liver or the heart ease the patient's condition not only by drawing blood but also by producing a revulsive effect through the neuroreflex mechanism, which was in his time pointed out by Zakharyin. Moreover, their application is very useful in the presence of a danger of thrombus formation, in thrombophlebitis and coronary thrombosis, since hirudin secreted by leeches contributes to a reduction in blood coagulation and prevents the formation of thrombi; the latter is favoured by a decelerated blood flow, thickening (inspissation) of the blood, and a change in the vessel walls.

Treatment of oedema. As a diuretic remedy for the treatment of heart disease in the stage of oedema Russian physicians F. Inozemtsev, F. Karell, and S. Botkin recommended the use of milk, which is given to patients in fractional doses totalling 1 litre during one or two days a week. For the same purpose, A. Yarotsky recommended to fast on certain days allowing for only 500 g of cottage cheese daily during a period of 2-3 days. In clinical practice, fasting days are also used, when the diet consists of fruit, compote, vegetable salads, potatoes, various berries, and sometimes 100-200 g of sugar which also produces a diuretic effect. In the third stage of circulatory disturbance when oedema develops not only from cardiac failure but also from hypoproteinaemia, the diet should consist of readily assimilated proteins, such as those contained in curds, eggs, meat, liver, etc.

Other diuretics applicable are urea which is given in a dose of 30-50 g per 24 hours (formula No. 51), novurit (formula No. 43), phonurit (formula No. 43*a*), and hypothiaside (formula No. 46*a*) in combination with potassium chloride (formula No. 54*a*); novurit may be administered in suppositories.

Several days before administration of mercury diuretics it is recommended to take ammonium chloride (formula No. 44) or calcium chloride (formula No. 54). Novurit is administered not more often than once in five days with a watch being kept over the urine. In case of the onset of haematuria (blood-stained urine) novurit therapy must be immediately discontinued. The drug is contraindicated for inflammatory processes in the renal gromeruli, since retention of mercury in the body is likely to cause a further exacerbation of the process and disturb the renal function. In congestion albuminuria novurit on the contrary reduces stasis in the kidneys, improves circulation of the blood and eliminates albuminuria.

Diet. In the stage of decompensation, when water and salt are retained in the body the intake of liquid and salt should be strictly limited. The daily intake of liquid and salt allowed is 800 ml and about 2 g respectively. With an insignificant oedema this requirement may not be followed so strictly, but with an extensive oedema it is absolutely indispensable. The diet must include milk, cottage cheese, sour cream, eggs, white bread, butter, vegetables, lean meat, and fresh river fish. Sugar, jam, fruit jelly, and fruit are very helpful. Food should be taken in small portions so as not to burden the stomach and intestine and cause flatulence.

In the absence of spontaneous stool, the intestine must be daily cleansed with enema, because the faecal masses and gases accumulated in the intestine in constipation interfere with the heart's action. In constipation, saline purgatives may be helpful, which, besides faecal masses, also serve to eliminate liquid from the body. A patient with a heart disease should be weighed every week. In addition, a special record is taken of the amount of liquid intake and urinary discharge with a view to ascertaining a reduction or growth of oedema.

Care. Special attention should be given to the bed, which should meet specific requirements in view of the fact that a patient with heart disease is liable to have bedsores due to a disordered peripheral circulation, with subsequent ulceration and infection (erysipelas). The bed-sheet should be smooth and without folds, and a rubber pad wrapped in cloth is placed under the sacrum. The patient's head should be propped up, for which several pillows are used; at the head of the bed a head-rest wrapped in a bed-sheet may be placed. In case of severe dyspnoea the patient should be seated in a soft armchair or on a bed. His head should be propped up with a pillow and a foot-stool provided for resting his feet.

It is necessary to take special care of the skin in view of its extreme liability to injury in oedema. The thinned skin of the patient becomes dry, and on the slightest scratch the oedematous fluid escapes and macerates the skin, thus providing favourable conditions for the propagation of bacteria. Especially dangerous is a streptococcal erysipelas, which in the course of a cardiac diseases sometimes becomes fatal. The skin should be painted with petrolatum and lanolin and in the case of chafing, powdered with talcum and starch.

The patient must be given not only physical but also mental rest. No noise or exciting talk is allowed. A deep and quiet sleep is necessary, and soporific drugs may be administered for insomnia.

Health resort therapy. Sufferers from heart disease in the stage of congestive heart failure are given medical treatment in a hospital or at home. Patients with 1st-degree circulatory disorder may be sent for treatment to a sanatorium. Of the Soviet health resorts which enjoy well-deserved fame Kislovodsk and Sochi-Matsesta merit special notice. In Kislovodsk patients are given narsan (mineral water containing sodium carbonate, sodium chloride, carbon dioxide, and hydrogen sulphide) baths 10-12 times per treatment course. The water temperature must be within a range of 34-30°C. The duration of each bath is 6-15 minutes.

In Sochi patients are treated with Matsesta (hydrogen sulphide) baths, each course of treatment consisting of 10-12 bathings. The water temperature is 36-34°C, the time of bathing 6-15 min. Both carbon dioxide and hydrogen sulphide baths are contraindicated in kidney affection. Treatment with Matsesta baths is prescribed for those patients who have joint diseases concurrent with a heart lesion.

Patients with 2nd-degree circulatory disorder may be given treatment also at specialized (cardiologic) sanatoria at their place of residence. Patients with 3rd-degree circulatory disorder are given treatment only in a hospital or at home if appropriate conditions are available.

DISEASES OF THE HEART MUSCLE

Diseases of the heart muscle (myocardium) occupy an important place in cardiac pathology. Considerable disturbances in the blood flow are nearly always associated with a lesion of the heart muscle and disturbance of its function. In respect of aetiology and pathogenesis as well as clinical course the following forms of affection of the heart muscle are known: myocarditis, myocardial sclerosis, myocardial dystrophy and pulmonary heart disease (cor pulmonale).

Myocarditis

Myocarditis is an inflammation of the heart muscle developing in various infectious diseases, such as, for example, rheumatic fever, diphtheria, enteric fever, typhus, scarlet fever, tonsillitis, malaria, septic diseases, etc.

Pathogenesis. The pathogenesis of myocarditis is variable and depends on its aetiolcgy. In purulent septic myocarditis the causal agents of the principal disease invade the heart muscle and produce an inflammation there. In many infectious diseases, inflammatory changes in the myocardium are apparently due not only to the action of microorganisms but also of a toxin, whereas in rheumatic myocarditis they are of an infectious and allergic character.

Pathologic anatomy. The pathoanatomic picture of myocarditis depends on the character of infection. In rheumatic fever, Aschoff-Talalayev's bodies form in the interstitial tissue of the myocardium and about the vessels. Affection of the muscle fibres is secondary, the muscles being replaced by scar tissue. In diphtheritic myocarditis, the muscle fibres undergo diffuse granular, fatty, and hyaline degeneration and are partly destroyed and later replaced by connective tissue. In myocarditis, irrespective of its aetiology, degenerative and necrotic processes occur in the muscle fibres and proliferative changes, in the interstitial tissue. There is a certain regular relationship between the character of morphologic changes and the clinical course of myocarditis. In predominance of degenerative and necrotic processes myocarditis runs a more severe course than when proliferative (cicatricial) changes prevail.

Clinical picture. The clinical picture of myocarditis depends on the degree of extension and course of the disease. In respect of its extension myocarditis may be focal or diffuse, and in respect of the course, acute and chronic. The disease takes a chronic course in a recurrent lesion of the myocardium which takes place, in particular, in rheumatic fever.

The mildest symptoms are displayed by focal myocarditis which is accompanied by tachycardia, less often by bradycardia, cardiac arrhythmia, more often by an extrasystole, a muffled first sound and a systolic muscular murmur at the heart apex.

Diffuse myocarditis runs a much more severe course. The patient complains of an oppressive sensation and pain in the heart, palpitation, and breathlessness. He has pallor of the skin, sometimes with cyanosis, and finds relief in a certain posture. The face is oedematous, the heart is considerably enlarged on both sides, and the heart sounds are dull, with a systolic murmur heard at the heart apex. The diastole is sometimes shortened, as a result of which the rhythm of cardiac contractions becomes pendulous in character.

The liver is congested, the cervical veins are engorged. There is fever, leucocytosis, anaemia and an elevated ESR. Electrocardiography shows a disturbance in impulse conduction, even disappearance of individual ventricular systoles. Upon recovery, all of the above-mentioned symptoms cease.

Rheumatic myocarditis runs a rather mild course. Severe disturbances of the blood flow due to myocarditis are seen in diphtheria when the slightest physical strain, even rising from bed, may cause sudden death. The course of myocarditis depends also on the condition of the vascular system; cases are frequent of death due to an acute vascular insufficiency in acute infectious diseases. When cardiac failure is joined by vascular insufficiency there is pallor of the skin, a rapid, soft and small pulse, and a fall in the arterial pressure.

Prognosis should be made with extreme caution, in account not only with functional disturbances in the cardiovascular system but also the course of the principal infectious disease responsible for myocarditis. The latter in most cases ends in complete recovery, and only very deep lesions of the heart muscle lead to the formation of scar tissue which later may give rise to persistent disturbances in conduction, block and other arrhythmias, as well as to a circulatory disorder.

Prophylaxis. Prophylaxis of myocarditis consists in measures to prevent infection which may cause myocarditis, timely and vigorous hospital treatment of infectious diseases (with salicylates in rheumatic fever, antidiphtheric serum in diphtheria, penicillin and other antibiotics in sepsis, etc.).

Treatment. In the case of a circulatory disorder the patient is given camphor (formula No. 31), caffeine (formula No. 33), corglycon and strophanthin (formulas Nos. 13, 12) with glucose, and strychnine (formula No. 37). Digitalis is contraindicated for a conduction disturbance. The diet must be sufficiently nourishing and rich in proteins and vitamins.

Cardiosclerosis

Sclerotic changes in the heart muscle (cardiosclerosis) result from infectious myocarditis (diphtheria, scarlet fever, typhoids), rheumatic myocarditis and atheromatosis of the coronary vessels.

In infectious and rheumatic myocarditis connective tissue proliferates in interstitial tissue and undergoes sclerosis. Atheromatosis of the coronary vessels disturbs the nutrition of the heart muscle by inadequate blood supply due to narrowing of the coronary vessels. This causes death of a part of the muscle fibres, their replacement by connective and scar tissue.

Clinical picture. Cardiosclerosis usually develops at an advanced age and produces no symptoms for a long time. Only upon a disturbance in myocardial function do signs of chronic or acute curculatory insufficiency become evident. Cardiac dullness increases because of dilatation of the heart and hypertrophy of the heart muscle. The cardiac sounds, particularly the first, are muffled. At the heart apex a systolic murmur is audible which is caused by relative insufficiency of the mitral valve or reduction in the tone of the heart muscle. As the disease progresses, there appear symptoms of heart failure—dyspnoea, palpitation, distressing sensations in the heart on the slightest physical strain, and oedema in the legs. Later these symptoms are present even at rest. There is growing oedema, venous stasis in the pulmonary circulation and liver, and ascitis. The urinary secretion is very scanty.

Cardiosclerosis is often accompanied by stenocardial pains in the heart, since it is sometimes concurrent with sclerosis of the coronary vessels predisposed to spastic contractions. In addition, there is often arrhythmia caused by cicatricial changes in the conducting system.

Treatment and prophylaxis. Treatment should be directed to eliminating the causes of cardiosclerosis and normalizing the deranged blood flow. Complete cure of developed cardiosclerosis is impossible in view of the presence of irreversible changes in the heart muscle. Therefore, one should content oneself with measures to prevent a further progression of cardiosclerosis. In rheumatic sclerosis measures to prevent relapses of rheumatic fever must be taken. For restoration of the disturbed blood flow, the patient is strictly enjoined to lead a hygienic way of life, with confinement to bed, moderate milk and vegetable diet without salted and spicy food, severe restriction on liquid intake, abstinence from tobacco and liquors. In cardio sclerosis of syphilitic aetiology, the patient is given bioquinol (formula No. 188), novarsenol (neoarsphenamine) (formula No. 189), in coronarosclerotic cardiosclerosis, iodine preparations for a prolonged period, such as potassium iodide (formula No. 190) or iodine tincture with potassium iodide (formula No. 176), euphyllin (aminophylline) (formula No. 26), rutin (formula No. 42*a*), methionine (formula No. 194), linethol (formula No. 195), and vitamins C and B_6 (pyridoxine—formulas Nos. 157*a*, 157*b*). For treatment of circulatory disturbance, see p. 229-30.

Myocardial Dystrophy (myocardiodystrophia)

Dystrophy denotes changes in the heart muscle which arise from a disturbance in the biochemical, physicochemical and biological energy processes. Dystrophy develops gradually on the basis of a disturbed nutrition of the heart muscle of variable origin. It is noted in all types of vitamin deficiency, particularly in B_1 avitaminosis (beri-beri), thyrotoxicosis, severe anaemia, intoxication (with carbon monoxide, phosgene, phosphorus, chloroform), in cachexia, and in intensified work of the heart.

Dystrophy of the heart muscle in vitamin B_1 deficiency results from a metabolic disorder associated with the latter. The muscle fibres become swollen, with a serous inflammation, which in time leads to the formation of a fine connective tissue. Upon adjustment of the dietary pattern all of the above-mentioned changes disappear and recovery is rapid.

The patient complains of dyspnoea, pain in the heart and liver. On objective examination, there is considerable dilatation of the heart, symptoms of congestion in the liver and the periphery.

The cardiac muscle in a thyrotoxic heart shows biochemical and later histopathologic changes. Cases are not infrequent, however, of only biochemical changes being discovered in the heart muscle. They result from a disturbance in tissue metabolism consequent to an intensification of oxidative processes and oxygen deficit. Tachycardia which is due to an enhanced excitability of the heart and direct action of thyrotoxin (thyroid gland secretion) on the heart muscle aggravates the disturbance of metabolic processes in the myocardium.

In far advanced cases of myocardial dystrophy the patient complains of breathlessness and palpitation even at rest. The heart is enlarged, with a systolic murmur heard at the heart apex and in the left second intercostal space, frequent cardiac arrhythmia, peripheral oedema and fluid effusion into the cavities, symptoms of stasis in the pulmonary circulation and the veins of the general circulation.

The pulse is frequent and quick, the systolic arterial pressure is heightened, and the diastolic pressure is normal. The pulse pressure is high. The patient dies of heart failure. It is often necessary to differentiate this disease from mitral valve insufficiency of rheumatic origin since in a heart affected by thyrotoxicosis the fibrous ring is weakened as a result of dilatation of the left ventricle and dystrophic changes in the myocardium; a relative insufficiency of the bicuspid valve is manifest. Excision of the thyroid gland in a patient with a thyrotoxicosis-affected heart brings great relief to the heart.

Severe anaemia also leads to dystrophic changes in the heart muscle with pains in the heart region (anaemic heart). The pains are anginose in character and are caused by oxygen deficiency, since the number of oxygen carriers—erythrocytes—is considerably reduced. In anaemic conditions, just as in obesity, there is often a fatty degeneration of the heart, when fat infiltrates between the muscle fibres. In severe chronic anaemia, particularly in pernicious anaemia, the heart is enlarged, its contractions are weak, the cardiac sounds are muffled, and the pulse is small. In addition to dyspnoea there is oedema which is partly due to protein deficit in the blood. Functional murmurs of anaemic origin are audible over all orifices which result from a decrease in the viscosity of the blood and reduction in the tone of the heart muscle.

In the treatment of dystrophy of the heart muscle, main attention must be paid to measures to strengthen the latter. In addition, steps must be taken to eliminate the causes, i.e., eliminate, for example, the factors responsible for anaemia. Of the other remedies, it is recommended to use raw liver, iron preparations (formulas Nos. 140, 136), and arsenic (formula No. 131), which often liquidate anaemia and along with it myocardial dystrophy.

Pulmonary Heart Disease (cor pulmonale)

Cor pulmonale is the name of those forms of cardiac failure which result from pulmonary diseases, in particular, emphysema, pulmonary fibrosis, fibroid tuberculosis as well as bronchial asthma, adhesive pleurisy, etc.

These diseases, in their final stages, lead to severe disturbances in circulation of the blood.

Pathogenesis. The development of cor pulmonale is due to emptiness and atrophy of a considerable part of the pulmonary capillaries and reduction in the vascular bed of the pulmonary circulation, which largely interfere with the circulation of blood within the lungs. The increased heart action due to hypertension in the pulmonary circulation causes hypertrophy and dilatation of the right ventricle analogous to changes in the left ventricle in the case of hypertension in the general circulation.

Clinical picture. The clinical picture varies with the stages of the disease. At the onset of the disease, in the period of pulmonary insufficiency, the patient complains of dyspnoea, cough and attacks of asphyxia. Cyanosis and dyspnoea may periodically aggravate in connection with progression of the disease process in the lungs and the development of asthmatic bronchitis and bronchospasm. The latter are rather quickly eliminated by administration of ephedrine (formula No. 30) and atropine (formula No. 35). In the period of cardiopulmonary insufficiency, inspection reveals an emphysematous thorax, diffuse cyanosis, engorged veins on the neck, extensive oedema of the extremities, ascitis, hydrothorax, orthopnoea, and clubbed fingers and toes. The right ventricle is dilated and hypertrophied. At the lower margin of the sternum there is a systolic murmur caused by a relative insufficiency of the tricuspid valve. There is cardiac dullness, tachycardia, systolic pulsation of the liver, and a positive venous pulse; the blood flow is decelerated.

The venous pressure is increased to 150-200 mm of water. At the same time, there is an insignificant increase in the arterial pressure. The urinary organs show signs of stasis in the kidneys—oliguria, albuminuria, and haematuria. The urinary sediment contains a considerable number of cylinders.

Because of massive albuminuria and hypertension, cor pulmonale is sometimes mistaken for nephritis. Changes in the lungs, however, and enlargement of the right ventricle are not characteristic of this disease. Cor pulmonale may sometimes be confused with a decompensated mitral defect. With a history of lung disease, chest deformity, clubbed fingers, purple-blue discoloration of the skin and absent signs of a mitral disease the diagnosis of a heart defect is untenable.

Treatment and prophylaxis. In the case of pulmonary insufficiency the prognosis is favourable. A change of the place of residence for a warmer climate, prevention of chills, as well as treatment of the principal disease responsible for pulmonary insufficiency, may check the further development of the disease process in the lungs. The prognosis is much worse in case of supervention of cardiac insufficiency. In the early stages the patient's condition may be relieved with restriction of liquid and salt intake, application of dehydrating drugs (novurit, phonurit, hypothiaside), euphyllin, oxygen therapy and drugs improving the heart's action.

DISTURBANCES IN THE HEART'S RHYTHM (ARRHYTHMIAS)

Disturbances in the heart's rhythm are due to various disorders in the conducting system of the heart which may be of a local or central origin. It is known that the heart's action depends on three factors—automatism, excitability and conduction.

Regular and periodic contraction of the heart is caused by stimuli which arise automatically in the right atrium at the orifice of the vena cava superior in Keith-Flack's node (sino-atrial node). From this node stimuli are conveyed to the Aschoff and Tawara node of located in the interatrial septum and further to the bundle of His which ramifies into two branches when passing through the fibrous ring of the heart. One of them leads to the right, the other to the left ventricle along the cardiac septum. Both branches of the bundle of His are continuous with the Purkinje fibres at their ends which transmit stimuli to the heart muscle and cause its contractions. The entire system through which stimuli are conveyed is called the conducting system and consists of embryonal muscle fibres.

The rhythmic action of the heart is controlled by the sympathetic nerve and the vagus nerve which innervate the heart. Stimulation of the vagus nerve decelerates the heart's rhythm, while stimulation of the sympathetic nerve produces the opposite effect. On the aorta, the pulmonary artery, and the coronary arteries of the heart the vagus nerve and the sympathetic nerve form plexuses ending with aggregations of nerve cells—ganglia which give off nerve branches to muscle fibres. The activity of the vagus and sympathetic nerves is regulated by the cerebral cortex. In addition to the above-mentioned two nerves, I. Pavlov discovered another nerve intensifying heart contractions through a trophic influence on the heart muscle, i.e., by improving the nutrition of the muscle so as to increase its vital capacity.

The rhythmic action of the heart is disturbed under the following conditions: (a) disturbance in the generation of stimuli, i.e., disturbance in automatism; (b) change in the normal excitability of the sympathetic and parasympathetic nerves; (c) disturbance in the conducting system of the heart, the sino-atrial node, the bundle of His and its branches; (d) change in the atrial and ventricular muscles.

Disturbance in Automatism

Disturbances in automatism, i.e., in the generation of stimuli, cause sinus tachycardia, sinus bradycardia and respiratory (juvenile) arrhythmia.

Sinus tachycardia. Tachycardia or acceleration of the pulse rate is a condition in which the number of heart beats reaches 100-200 per minute, while stimuli for such heart contractions arise at their usual site, i.e., in the sino-atrial node. It is encountered in physical overexertion, fever, intoxication, caffeine, nicotine, and atropine poisoning, etc. Acceleration of cardiac contractions to 120-130 beats per min is a sign of a serious affection of the heart.

Sinus bradycardia. Bradycardia or slowness of the pulse is a condition in which the rate of heart contractions decreases to 40-50 beats per minute. It is encountered with prevalence of the vagus nerve tone and is subdivided into the following groups: (a) bradycardia resulting from stimulation of the vagus nerve centre in case of an increased intracranial pressure (tumours, meningitis, cerebral dropsy); (b) bradycardia resulting from stimulation of the vagus nerve by pressure from a tumour, pleural adhesions, lymph nodes; (c) reflex bradycardia resulting from diseases of the gallbladder, peritonitis, meteorism, etc. Bradycardia may be due to sclerotic changes in the sino-atrial node itself but it also may be encountered in perfectly normal persons engaging in physical work as well as in well-trained athletes.

Respiratory arrhythmia is otherwise known as juvenile since it is encountered as a physiologic phenomenon in childhood and adolescence if the tone of the nervous system has not yet developed stability. It also depends on the instability of the vagus nerve tone. It essentially consists in acceleration of the pulse during inspiration and deceleration during expiration. Owing to this the heart contractions become arrhythmic but this arrhythmia has no negative influence on the heart's action.

In adult persons, respiratory arrhythmia is secondary to a disorder in the central nervous system, when even such slight stimulation as a deep inspiration increases the tone of the vagus nerve which produces an inhibitory influence on the sino-atrial node. Respiratory arrhythmia may be notable in extremely emaciated persons and in convalescence.

Respiratory arrhythmia may be relieved by measures to increase the activity of the sympathetic nerve or reduce the tone of the vagus nerve. This may be achieved by exercise and administration of atropine. It also disappears in strong emotions and febrile conditions.

Disturbance in Excitability of the Conducting System

Extrasystolic arrhythmia (arhythmia extrasystolica). This is one of the disorders of the excitability of the conducting system. Normally, stimuli for heart contractions arise in the sino-atrial node and, passing through the conducting system, cause contractions of the atria and then of the ventricles at perfectly regular intervals. Extrasystolic arrhythmia is characterized by the appearance of additional, irregular or premature contractions of the heart. Under normal physiologic conditions, premature stimuli may cause contraction of the heart if it has passed the absolutely refractory phase and entered the relatively refractory phase. For the heart contractions to occur in relatively refractory phase, however, the stimuli must be considerably stronger than normally. With a number of pathologic changes in the heart, conditions required for the production of unusually strong stimuli are created.

In respect of their site of origin, extrasystoles are subdivided into sinusal, atrial, atrioventricular and ventricular. Physico-chemical, morphologic and functional neuroreflex changes occur in the abovementioned places which make it possible to generate unusually strong stimuli. Morphologic changes may result from inflammatory and sclerotic processes in the heart. Functional disorders are caused by a disturbance in the central nervous system or reflex influences from pathologically changed abdominal organs. Extrasystoles arising from disorders in the vegetative nervous system are subdivided into vagal and sympathetic. Neuroreflex extrasystoles are more frequently noted in young persons in the period of pubescence, in smokers, in excitable persons, as well as in cases of abnormality in some visceral organs—the gallbladder, the stomach, the intestine, the vermiform appendix, etc.

The diagnosis of extrasystolic arrhythmia is based on consideration of the patient's complaints, auscultation of the heart, examination of the pulse and analysis of electrocardiographic findings. The patient feels an extrasystole as an irregularity in the heart beat, a sensation of sinking into emptiness. Sometimes the patient may not be conscious of an extrasystole.

On auscultation of the heart, in arrhythmia the diastole arriving after an extrasystole is more prolonged than normally (compensatory pause). The regular stimulus coming from the sino-atrial node does not cause contraction of the heart muscle, since the latter, after spending all its energy in the extrasystolic period, becomes resistant to excitation, i.e., refractory. The compensatory pause is especially marked in ventricular extrasystole.

Additional stimuli coming from a point lying outside of the sino-atrial node sometimes occur in a definite sequence—after 1, 2, 3, or 4 normal stimuli. In such cases palpation of the pulse may reveal an additional pulse wave occurring after 1, 2, 3, or 4 normal beats. Where every normal heart contraction is followed by an additional pulse wave the so-called begeminal pulse (pulsus begeminus) is present. A pulse with an additional wave after two normal waves is called pulsus trigeminus; that with the appearance of an additional wave after three normal waves is called pulsus quadrigeminus. On examination of the pulse extrasystoles are felt as additional beats to the stronger normal pulse waves. The pause before an extrasystole is shorter and after it, longer than normally. The appearance of extrasystoles after equal time intervals, i.e., after equal numbers of normal heart contractions is called allorhythmia.

Extrasystoles resulting from morphologic and inflammatory changes in the heart muscle require treatment of the principal disease which has produced the latter. Functional extrasystoles may be quickly eliminated by measures to improve the patient's general health—aquatic procedures, exercise, sports, etc. Patients are recommended to take Bechterew's mixture, small doses of luminal, novocaine amide (formula No. 19), etc.

Atrial fibrillation. This condition is characterized by a disturbance in the rhythm of the heart contractions caused by an increase in the excitability of the atrial muscle and a decrease in stimuli conveyance through the conducting system. These changes occur in distention of the atrial walls and the resultant dystrophic changes in the myocardium which take place in stenosis of the left atrioventricular orifice, atherosclerotic cardiosclerosis, myocardial infarction and thyrotoxicosis.

In various parts of the atrial muscle there arise numerous stimuli which are not conveyed over a distance but die down on the spot, producing only isolated contractions of muscular fibrils in the form of flutter. Only isolated stimuli reach the Aschoff and Tawara node and cause arrhythmic contractions of the ventricles. The following three forms of atrial fibrillation are known: (a) atrial fibrillation with stimuli arising in the atrial muscle at a rate of 350 per minute;

(b) atrial fibrillation with stimuli occurring at a rate of 300 to 600 per minute, and (c) paroxysmal form of atrial fibrillation.

In atrial fibrillation, the stimuli rhythmically arrive at the Aschoff and Tawara node, but not all of them reach the ventricles and consequently do not cause their contraction, since the atrioventricular node sends to the ventricles not more than 180-200 stimuli per minute.

In atrial fibrillation when the number of atrial stimuli reaches 600, no complete atrial contractions take place; there is only contraction of isolated fibrils, and the ventricles are reached only by certain irregular stimuli, owing to which the ventricles contract arrhythmically. With regard to the number of cardiac contractions, atrial fibrillation may be as follows: (a) bradysystolic, with up to 80 contractions per minute; (b) tachysystolic, with over 100 contractions per minute.

The paroxysmal form of atrial fibrillation is encountered in disorders of the nervous system and occurs periodically as attacks of paroxysmal tachycardia which soon passes.

When identifying the form of atrial fibrillation one must not be guided by the number of pulse waves, since some stimuli arising in the beginning of a diastole produce such a weak contraction of the heart that the semulunar valves fail to open and the blood is not supplied into the aorta. In such cases, on counting heart contractions and the pulse one will see that their figures do not coincide, i.e., that a pulse deficit is present. A reduction in pulse deficit is evidence of general amelioration, and vice versa.

Paroxysmal tachycardia manifests itself by a sudden onset of palpitation and a sensation of a heart thrust in the beginning and at the end of an attack. It comes to an end just as suddenly. Tachycardia is encountered in heart defects, myocardial infarction, cardiosclerosis, as well as in functional disorders of the nervous system. The rate of cardiac contractions reaches 180-200 beats per minute, with a normal rhythm. It is impossible to count the pulse.

An attack of tachycardia is accompanied by a feeling of weariness, creeps all over the body, dizziness, and chilling and numbness of the extremities. There is a cold sticky perspiration, swelling and intense pulsation of the veins on the neck, and continuous quivering in the epigastrium and at the heart apex. Both cardiac sounds are equally sonorous, with equal intervals between them, i.e., they become embryocardiac in character. The arterial pressure is diminished.

The supply of blood to the coronary vessels is sharply reduced due to a short diastolic period, which impairs the conditions for nutrition of the heart muscle. The reduction in the contractile function of the heart muscle is also due to the fact that the diastole is too short for the heart muscle to be able to restore its function in full. In addition, there is a blood deficit in the ventricles. All this causes a reduction in the systolic and minute cardiac output.

Attacks may recur several times during a day, sometimes they occur seldom, once a year and even rarer. In those cases when an attack continues for several days there is a disturbance in circulation of the blood with cyanosis, dyspnoea, reduced diuresis, symptoms of stasis in the pulmonary circulation and in the veins of the general circuit, and oedema of the face and extremities. The course of an attack depends on the character of paroxysmal tachycardia. Attacks, including prolonged ones, associated with functional disturbances in the nervous system run a favourable course. Paroxysmal tachycardia of an organic character runs a severe course, with a considerable circulatory failure, and sometimes ends in death.

Treatment primarily consists in administration of drugs reducing the tone of the sympathetic nervous system, since the latter's overexcitation plays an important role in the development of an attack. For this purpose, the patient is advised to hold his breath, make a deep inspiration, change the position of the body, and turn the head to the side. Relief is sometimes produced by pressure for 10-12 seconds on the carotid sinus which lies at the level of the upper margin of the thyroid cartilage inwardly to the sternocleidomastoid muscle. An attack may be stopped also by pressure applied to the eyeball, and by administration of a hot or cold foot bath which stimulates the skin.

Drug therapy consists in intravenous infusions of a strophanthin solution, peroral administration of quinidine hydrochloride or quinidine sulphate in a dose of 0.1-0.2 g three times daily or, which is better still, a 50 per cent solution of chinini bimuriatici in a dose of 1 ml intramuscularly to reduce the excitability of the heart muscle. Among the ganglioblocking drugs procain amide is efficacious (novocaine amide) in a dose of 0.5-1 g per administration, the total daily dose being up to 4 g; it may also be administered in the form of a 10 per cent solution intramuscularly in a dose of 5-10 ml or intravenously in a dose of 2-5 ml in case of emergency. For prolonged attacks with a circulatory disorder, the patient is given injections of caffeine, camphor in a dose of 10 ml, a 10 per cent sodium bromide solution and also strophanthin with glucose.

In the period between attacks it is recommended to carry out treatment of the principal disease responsible for paroxysmal tachycardia. In case of predominance of functional disturbances in the nervous system sponge baths, saline-pine baths, physical exercises, and psychotherapy are prescribed.

Heart Block

In a disturbance of the normal conduction of the stimulus from the atria to the ventricles or along a branch of the bundle of His and its ramifications in the myocardium the heart's action becomes arrhythmic (heart block). Disturbance in conduction results from organic lesions (rheumatic granuloma, scars left by infarctions, cardiosclerosis, syphilitic gumma) or reversible functional (dystrophic) changes. Four degrees of heart block are known.

In first degree block there is only a certain prolongation of the time a stimulus traverses from the atria to the ventricles, which is manifested only by an increase in the *P-Q* interval of an organic or functional character, the latter, as distinct from an organic disturbance, being eliminated by an injection of atropine.

Second degree block is notable in cases where conduction of the atrial stimulus is considerably impeded. Part of the stimuli pass through the node of Aschoff and Tawara normally, while the time conveyance of the subsequent stimuli is gradually lengthened because of a decrease in the excitability of the atrioventricular node to such a degree that one of the regular stimuli fails to cause excitation, which results in failure of ventricular contraction. The second degree block is determined from an electrocardiogram as well as from the pulse as a result of a periodic dropping out of every 5th-7th contraction of the heart. The second degree block is also known as Wenckebach-Samoilov's periods, named after the authors who were the first to describe this type of conduction disturbance.

Third degree conduction disturbance is otherwise known as partial atrioventricular block. It develops in case of severe disturbances in the node of Aschoff and Tawara as a result of which the ventricles are reached by every third or forth stimulus conveyed from the atria, so the pulse becomes slow.

Fourth degree block or transverse complete block is encountered in complete disruption of atrioventricular conduction resulting from organic (granuloma, gumma, scar, knife or bullet wound) or functional lesions.

In transverse or complete block the atria and ventricles work independently and have their own rhythms. The number of cardiac contractions depends on the site of anatomic damage. The larger the distance between it and the node of Aschoff and Tawara, the less the number of cardiac contractions. A lesion of the bundle of His at the site of its exit from the atrioventricular node results in 50 contractions per minute. Ventricular contractions in transverse block are due to the fact that any point of the heart muscle itself or the conducting system may become the site of independent excitation. Gradual development of transverse heart block remains unnoticed by the patient since his body becomes adapted to a reduced number of cardiac contractions, even to 40-30 beats per minute.

A sudden onset of transverse block, however, may cause an attack resembling epilepsy. There is severe dizziness, sometimes with a brief loss of consciousness, convulsions with biting of the tongue and foaming at the mouth, and involuntary urination and faecal discharge. The above-mentioned clinical manifestations which are known as Adams-Stokes-Morgagni's syndrome result from a brief cessation in blood supply to the brain. In the period between attacks, patients with complete block retain their working capacity if no circulatory failure is present. In a disturbance of circulation of the blood, in addition to the slow pulse, there is enlargement of the heart. On ausculation of the heart, there are rare, somewhat muffled sounds, the first sound being periodically intensified (cannon sound, according to N. Strazhesko), as a result of a simultaneous contraction of the atria and ventricles. Transverse block caused by an organic lesion is constant in character. Transverse block due to a functional change in the conducting system is replaced by a normal cardiac rhythm.

Sometimes, under the same conditions as in transverse block, there is *block of a branch of the bundle of His*, i.e., interruption in stimuli conduction along one of the bundle branches. In such cases, the stimulus normally reaches the heart muscle of one of the ventricles through an intact branch from where it is conveyed through the heart muscle to the ventricle with the damaged bundle branch. As a result, the ventricles contract asynchronously as is evidenced by an electrocardiogram.

Treatment. Drugs are administered to cure the principal disease responsible for transverse block: in syphilis, bioquinol and salvarsan; in sclerosis, iodine preparations, etc. In disturbance of cardiac function the patient is given Adonis vernalis per os and glucose intravenously. Digitalis is contraindicated.

DISEASES OF THE PERICARDIUM

Pericarditis occurs with a number of infectious diseases—enteric fever, smallpox, tularaemia, brucellosis, and most frequently with rheumatic fever and tuberculosis. It is encountered in diseases of the lungs and the mediastinum—pneumonia, pulmonary cancer, thoracic injuries, etc. Pericarditis also results from general toxaemia which occurs in uraemia and myocardial infarction, when the inflammatory process extends as far as the outer sac.

Pericarditis is found in two varieties: (a) fibrinous, or dry, and (b) with effusion, or wet. Pericarditis with effusion, or wet pericarditis, is in its turn subdivided into serous, purulent and haemorrhagic. The inflammatory process is followed by complete resorption of the effusion or the formation of adhesions, and sometimes complete adhesion of the inner surface of the pericardium with the heart, with obliteration of the pericardial cavity. The thickened pericardium shows the presence of lime deposits. This outcome of pericarditis is known as the armoured heart. Sometimes, the pericardium becomes adherent to the surrounding organs.

Dry Pericarditis (pericarditis sicca)

The clinical manifestations of dry pericarditis are quite insignificant. The patient complains of slight pains in the heart region. Among the objective symptoms, the most important is the pericardial friction sound, which is audible both during a systole and during a diastole over a circumscribed area, most often near the sternal border in the third and fourth intercostal spaces. The pericardial friction sound is augmented on pressure with a stethoscope, and is better audible when the patient is standing. This sound is due to the formation of fibrin deposits on the pericardial surfaces rubbing each other. It may be gentle or scratching, is sometimes felt by the hand, persists for 1-3 days and disappears upon the effusion of exudate which separates the pericardial surfaces from one another. Dry pericarditis may end in recovery in 2-3 weeks' time or become wet.

All types of acute pericarditis, except for the uraemic type, are accompanied by fever and leucocytosis. The other symptoms encountered in pericarditis are due to the principal process responsible for this disease. Identification of pericarditis is not difficult. In order that the pleural friction sound may be differentiated from the pericardial sound, one should first of all determine the character of this sound: the pleural friction sound is more diffuse, better audible in the inspiratory phase, and absent when breathing is stopped.

Prognosis depends on the course of the principal disease.

Pains are relieved by the application of mustard plasters, iodine tincture painting, aspirin, pyramidon, and narcotics—dionine, pantopon (formulas Nos. 103, 107). In rheumatic pericarditis large doses of salicylates—from 8 to 10 g in 24 hours—are prescribed, in pneumonic pericarditis, sulpha drugs and penicillin; in pericarditis of tuberculous origin, streptomycin and phthivazide.

Pericarditis with Effusion (pericarditis exudativa)

Pericarditis with effusion of serofibrinous character is most commonly encountered in rheumatic fever, tuberculosis and pneumonia; in septic processes, the exudate becomes purulent. In tuberculous pericarditis, lymphocytes prevail in the exudate; in septic processes, neutrophils. Perforations of the oesophagus, thoracic injuries, and rupture of a subdiaphragmatic abscess cause putrid pericarditis. Haemorrhagic pericarditis most commonly occurs with malignant tumours of the mediastinum. The amount of exudate in the pericardial sac may vary from 200 to 2,000 ml. The gravity of the process depends on accumulation of fluid in the pericardial cavity, which entails an increase in the intrapericardial pressure, making it very difficult for the blood to fill the ventricles during a diastole and also interfering with the drainage of blood from the venae cava. The course of the disease is influenced by neuroreflex

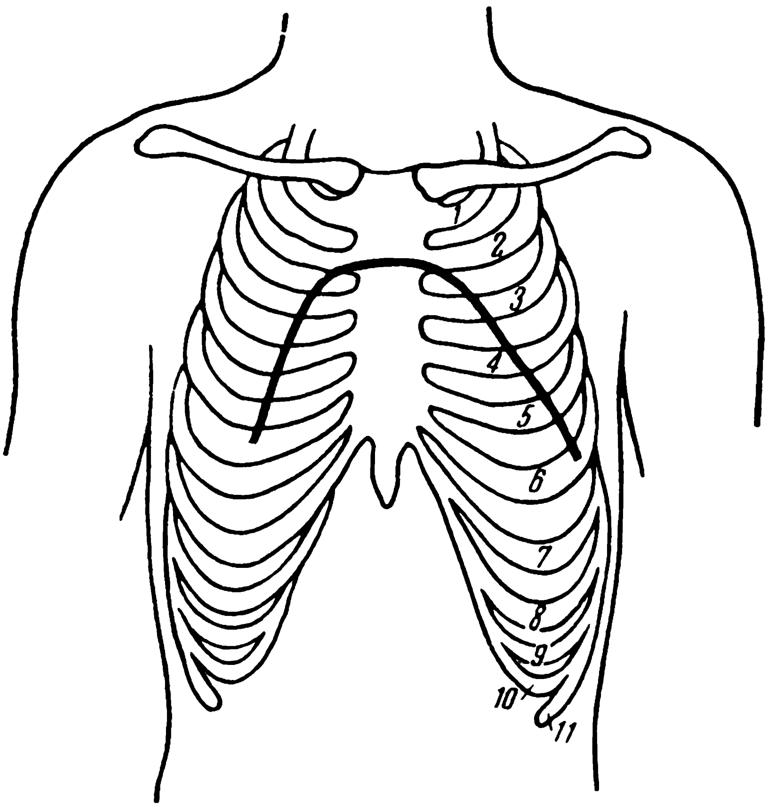

Fig. 43. Heart borders in pericarditis with effusion

stimuli conveyed from the receptor fields of the pericardium and from the adjacent organs.

The patient's condition is very grave. He lies on his back or takes a sitting or a knee-elbow position. The characteristic symptoms of pericarditis are pallor or ash-grey colour of the face, cold perspiration and a feeling of heaviness, and sometimes intense compressing pains in the heart radiating to the neck, stomach and back.

In profuse exudation, the entire pericardial region bulges, while the intercostal spaces are flattened. The apex beat and heart pulsations are absent. The liver, particularly its left lobe, protrudes from under the costal arch. All borders of cardiac dullness are widened, and relative dullness is absent (Fig. 43), while the heart sounds are quite muffled. The pulse is rapid, sometimes arrhythmic.

Large effusions cause compression of the mediastinal organs. This is manifested by puffiness of the face and pelerine-shaped oedema, a husky and toneless voice, dysphagia as a result of compression of the oesophagus or the vagus nerve, cough due to compression of the bronchi and trachea, hiccough resulting from pressure on the diaphragmatic nerve, and angina pectoris owing to compression of the coronary vessels.

Rheumatic pericarditis with effusion undergoes resorption in time. In tuberculous pericarditis, resorption is noted less frequently. After abatement of acute symptoms, the disease most commonly takes a chronic course, with caseation of the pericardial surfaces.

The x-ray picture is quite typical and facilitates diagnosis: the heart acquires a triangular shape and all arches are flattened.

Pericarditis differs from the ox heart (cor bovinum) in that the latter involves excessive distention of the heart and weakening of the apex beat. In pericarditis the area of dullness does not extend downwards and leftwards, as is the case in aortic valve insufficiency. As distinct from right ventricular insufficiency, pericarditis is not accompanied by pulsation of the veins of the neck. Pericarditis differs from left-side pleurisy in that in pleurisy the area of dullness is found both at the back and front on one and the same level, whereas in pericarditis the area of dullness at the back is located lower than at the front. In pleurisy x-rays distinctly reveal pulsation of the heart even if the latter is displaced. Sharp pains in pericarditis may give cause for an erroneous diagnosis of myocardial infarction; it should be remembered, however, that in pericarditis the sitting posture relieves pains, which is not the case in myocardial infarction.

In the acute stage of rheumatic pericarditis, sodium salicylate in a dose of 8-10 g or pyramidon in a dose of 2-3 g per 24 hours is prescribed; in septic pericarditis, penicillin in a dose of 800,000 units per 24 hours, and sulfa drugs. In purulent effusion evacuation of pus is indicated with subsequent injection of penicillin and other antibiotics into the pericardial cavity. Evacuation of the effusion is undertaken only in the presence of vital indications, when symptoms of cardiac insufficiency aggravate. The site of puncture is at the left margin of cardiopericardial dullness.

In the acute stage of the disease, pains are abated by the application of iodine, leeches, and mustard plasters to the heart region, as well as by narcotic drugs—pantopon, morphine. The patient should be calmed. For this purpose bromide (formula No. 114), tincture of valerian, and chloral hydrate (formula No. 120) are prescribed. Sometimes pain is assuaged by means of an intravenous injection of a 0.25 per cent novocain solution in a dose of 5-10 ml. Novocain anaesthesia of the most sensitive parts of the skin is also helpful.

Adhesive Pericarditis (pericarditis adhaesiva)

Acute pericarditis with effusion may terminate in adhesive pericarditis. Insignificant adhesions do not trouble the patient and are discoverable only at autopsy. In complete adhesive closure of the pericardial cavity the heart is enclosed within adhesions, in which lime is deposited; the heart seems to be covered with armour, from which the term "armoured" heart takes its origin. In some cases, adhesions fix the heart to the thorax and the mediastinum.

In adhesive pericarditis the heart is small in size, enclosed and covered with adhesions; there is a marked venous stasis in the liver and in the system of the portal vein. The patient complains of a feeling of heaviness in the epigastrium, which is due to stasis in the liver. The apex beat is absent or fixed, and is not displaced when the patient's position is changed. In adhesion of the pericardium to the front chest wall, the latter is indrawn during a systole and bulges during a diastole (paradoxical apex beat). Disturbance in the blood circulation is caused by difficulty in the diastolic filling of the heart and disturbance of the systolic ejection of blood into the aorta. The pulse is usually small, the pulse pressure and the arterial pressure are low; at maximum inspiration the pulse becomes smaller or disappears completely, although the cardiac tones are clear; therefore, the pulse is considered paradoxical. The liver is enlarged, the veins of the neck are swollen, and the venous pressure is considerably elevated. In cases where the adhesions are located in the region of the inferior vena cava, there is only enlargement of the liver; if, however, the adhesions are selectively located in the region of the superior vena cava, the liver is normal but the face is pasty. With adhesions in the region of the left atrium, there are symptoms of pulmonary congestion, which do not respond to digitalis and confinement to bed because they are caused by mechanical factors.

The presence of inconsistency between the small size of the heart and considerable congestive symptoms is important for identifying adhesive pericarditis; the condition is also indicated by persistent ascitis developing insidiously, without precedent rheumatic fever or heart defect, and resisting therapy. Therefore, adhesive pericarditis is often diagnosed as peritonitis or liver cirrhosis, from which this disease takes its name of pseudocirrhosis of the liver. Actually, in adhesive pericarditis, there is cirrhosis of the liver which results from stasis of the blood within it.

The only reliable method of treatment is surgical interference, which consists in removal of the scars binding the heart. Repeated punctures of the abdominal cavity and withdrawal of fluid, the application of diuretics and digitalis only temporarily ease the patient's condition.

ATHEROSCLEROSIS

Atherosclerosis affects the inner coat of an artery resulting in lipoid degeneration followed by the development of connective tissue (sclerosis). This disease occurs mainly in elderly and old persons, but may also be found at a young age. Atherosclerotic changes in the vessels are not always uniformly distributed. Most commonly, the disease process is localized mainly in the aorta, the coronary, cerebral, renal and mesenteric arteries, as well as in arteries of the extremities. Simultaneous atherosclerosis of the cerebral, cardiac and renal arteries also occurs frequently, the disease process being the most intense in one of the above systems.

Aetiology and pathogenesis. The aetiology of atherosclerosis is uncertain. Most probably, the principal causes of atherosclerosis are metabolic disorders, a deranged nervous regulation of the vessel wall, and a hyposecretion of the endocrine glands. Atherosclerosis develops mainly in persons eating protein and fatty foods to excess, leading a sedentary way of life, and abusing alcoholic drinks and tobacco. Another evidence in favour of metabolic disorders as the causal factor of atherosclerosis is the fact that in persons engaging in physical work and sports this disease is not frequent. There is also an undoubted connection between atherosclerosis and endocrinic changes in the body. This is confirmed by women being less susceptible to atherosclerosis than men, possibly because of the increased function of the thyroid gland in the former. Another important cause of atherosclerosis is spastic constrictions of blood vessels, which is evidenced by frequent concurrence of hypertensive vascular disease with atherosclerosis.

The essence of morphologic changes in arteries during atherosclerosis consists in deposition of cholesterol in their walls (lipoid infiltration). In the opinion of most contemporary authors, lipoid infiltration, rather than being caused by an absolute increase in the blood cholesterol level, is due to a decrease in the level of lecithin, which produces a change in the normal cholesterol-lecithin ratio. Cholesterol, which is normally retained in the blood by lecithin, falls out from a suspension and is deposited in the arterial intima.

Hypercholesterolaemia may be due to exogenous and endogenous factors. Among the exogenous factors is a diet with excess cholesterol (animal fats, caviar, brains, liver, etc.). Cases are frequent, however, when people eating exclusively fatty foods do not suffer from atherosclerosis or develop it not more frequently than vegetarians. The development of atherosclerosis cannot be fully explained by a disturbance in the cholesterol-lecithin balance and the resulting deposition of cholesterol in the inner coat of arteries. Most probably, a certain role is played by the state of the arterial intima itself. It is not unlikely that for cholesterol to become deposited, the inner coat must be damaged by dystrophic processes.

Great significance in the development of atherosclerosis is attached to an increase in the blood of the protein globulin fraction, as well as to lipoproteins (combination of cholesterol with globulins).

Arteries affected by atherosclerosis are indurated, lose elasticity and become narrower, which interferes with blood supply to organs. This leads to a nutritional disturbance and atrophic changes in the vitally important organs—the heart, the brain and the kidneys. In addition, in atherosclerosis the blood vessels are prone to spasms, which are accompanied by various symptoms depending on the predominant localization of the disease process: dizziness, angina pectoris, numbness of the extremities, etc.

Pathologic anatomy. In the inner coat of arteries, deposition of cholesterol and its esters is followed by formation of connective tissue and thickenings visible as plaques. The latter disintegrate and form a gruel-like (from Greek *athera*—gruel) fatty mass. This mass contains lumps of lime, cholesterol, fatty acids and protein substances. Sometimes the site of decomposed plaques undergoes ulceration. These plaques are of a yellowish colour and show the growth of connective tissue and deposition of lime, which causes the arteries to lose their elasticity. In the arteries of the heart, brain, kidneys and extremities the plaques cause constriction of the lumen to the degree of its complete obstruction. Because of the inadequate blood supply through the stenosed arteries dystrophy of the myocardial fibres develops followed by their sclerosis. Atheromatous plaques also form in the larger vessels—the aorta, the innominate, subclavian and iliac arteries—narrowing their lumina, though at places these vessels undergo distention due to the death of elastic tissue.

Clinical picture. Atherosclerosis is usually subdivided into the preclinical and clinical periods. The preclinical period of the disease runs a latent course. Usually, the patient gives no complaints except for nervous unrest. In the blood only biochemical changes are found. In particular, there is an increase in the level of cholesterol and lipoproteins in the blood and a change in the cholesterol-lecithin ratio. Detection of the disease in its preclinical period is very important since prophylactic measures are especially effective in this period.

Depending on manifestations of atherosclerosis the clinical period is subdivided into three stages: (1) the ischemic stage, with constriction of vessels, which leads to disturbance of function in the corresponding organs and dystrophic changes within them; (2) the thrombonecrotic stage, with the development of micro- or macrofocal necroses, with or without vascular thrombosis; (3) the sclerotic (fibrous) stage, with atrophy of the parenchyma and formation of scar tissue within the organs.

Irrespective of the stage of the clinical period, a patient gives characteristic complains and shows objective changes typical of atherosclerosis. There is pain and heaviness in the head, dizziness, tinnitus, pains in the heart, deterioration of memory and hearing, weakness in the legs, sometimes of the type of intermittent limping and insomnia. On inspection the patient with general atherosclerosis is found to have lusterless eyes, early greyness of the hair, loss of skin lustre and atrophy of the skin. Under the skin are visible large tortuous veins, tense tortuous pulsating peripheral arteries with hard walls. This is especially characteristic of the radial artery.

In atherosclerosis of arteries of the lower extremities there are intense pains in the toes and the gastrocnemius muscles, which become especially intense in walking, a feeling of numbness, and the creeps. The pain in the toes may become so severe as to make the patient frequently stop when walking. In complete closure of the lumen of an artery the pulse on the dorsal artery of the foot or on the posterior tibial artery is undetectable on palpation, and the extremity soon grows pale when raised. Sometimes, a trophic disturbance in the lower extremities leads to necrosis of the toes, i.e., gangrene.

Examination of the heart reveals hypertrophy of the left ventricle, a prominent apex beat, dull heart sounds, and a systolic murmur at the apex of the heart and over the aorta. A trophic disturbance in the heart muscle produces cardiosclerosis which for a long time may remain symptomless. There may be serious complications, however, such as attacks of angina pectoris, myocardial infarction, a paroxysm of cardiac asthma, a circulatory disturbance, etc.

In atherosclerosis of the aorta the increase in its size leads to approximation of its arch to the upper aperture of the thorax. In such cases, pulsation of the aorta may be felt in the jugular fossa behind the manubrium of the sternum. On auscultation, a systolic murmur is audible in the right second intercostal space, which becomes more intense when the patient's hands are raised (Sirotinin-Kukoverov's symptom). The second sound over the aorta is accentuated and has a metallic quality. X-ray examination shows elongation and distention of the aorta. The patient complains of chest pain radiating to the left arm. Apparently, this pain is associated with concurrent atherosclerosis of the coronary arteries or with excitation of the sympathetic ganglia situated on the aorta.

Sometimes an atherosclerotic patient feels an intense pain in the abdomen. At the same time, the abdominal aorta is hard and pulsating, while a systolic murmur is audible on pressure with the stethoscope. In atherosclerosis of the mesenteric vessels there may be a paroxysm of intense pain in the stomach, the so-called angina abdominalis, as well as thrombus formation in the lumen of these arteries followed by gangrene of portions of the intestine.

Atherosclerotic changes in the brain vessels cause dizziness, tinnitus, tearfulness, early fatigue on physical and mental exertion, syncope, and deterioration of memory (the patient has clear memories of what happened 10-15 years ago and is forgetful of recent events). Considerable atherosclerotic changes in the brain vessels may give rise to cerebral haemorrhage or vascular thrombosis with resulting paralysis, total or partial loss of speech due to the formation of foci of softened tissue at the sites of affection. The patient is prone to weep. Weakness of mind (dementia) sometimes develops and progresses to the degree of complete degradation of personality.

In atherosclerosis, considerable metabolic changes are noted. This refers above all to lipids and proteins in the blood serum. There is an increase in the cholesterol content of the blood, which is especially demonstrable in the case of a simultaneous increase in the amount of phospholipids and lipoproteins, α- and β-lipoproteins in particular. Cholesterolaemia alone cannot be a diagnostic sign, because distinct signs of atherosclerosis are often found with a normal amount of cholesterol, or, conversely, in hypercholesterolaemia complaints characteristic of atherosclerosis may be absent. Apparently, the cholesterol-lecithin ratio is an important factor in this case. An increase in the lecithin-cholesterol index above 1 is a favourable sign. In atherosclerosis, there are also changes in the blood protein fractions, particularly an increase in α_2-globulins and a reduction in albumins.

Differential diagnosis. Diagnosis of atherosclerosis is difficult in the preclinical, latent period because at this time atherosclerosis is manifested only by functional disturbances, and may be confused with neurosis of the heart, hypertensive vascular disease in the 1st stage, etc. In the clinical period of the disease a differential diagnosis must be established between atherosclerosis and rheumatic and syphilitic affection of the heart, myocardial dystrophy, hypertensive vascular disease, affection of the kidneys, and disturbance in the metabolic and endocrinic processes.

Prognosis depends on the extension, localization and advancement of atherosclerosis. In the initial stage, atherosclerosis impairs working capacity only slightly. Later, as the heart's action is weakened and the cerebral and sometimes renal blood circulation is deranged, the capacity for work is reduced considerably.

Prophylaxis and treatment. The development of atherosclerosis may be prevented by sports, physical culture, normal sleep, abstinence from smoking and alcohol, and a rational diet. One of the major factors responsible for atherosclerosis is overeating, particularly after 40 years of age. In medical treatment, just as in prophylaxis, major attention should be given to a therapeutic diet. Food must be variable in composition, and contain a sufficient supply of proteins (100 g daily), vitamins, fruit, vegetables, and lipotropic products.

The dietary patterns to be followed so as to prevent atherosclerosis include vegetable fats with highly unsaturated fatty acids entering into their composition (25-30 g daily), ascorbic acid (vitamin C), pyridoxin (vitamin B_6), choline, methionine, as well as products containing lipotropic substances: cottage cheese, well-soaked herring, cod-fish, cauliflower, oatmeal, yeast, etc. Other recommended foods are lean grades of meat and fish, eggs (not more than 4-5 eggs per week), vegetables and fruit. The intake of fats should not exceed 40-50 g and that of carbohydrates, 300 g daily. Smoked foods, pickles, salted foods, and tinned foods are forbidden. The intake of common salt and liquid, particularly in predisposition to arterial pressure increase and oedema, must be restricted.

Of medical drugs vitamins C and B_6 are prescribed in a daily dose of up to 100 mg, vitamin B_{12} in a daily dose of 100 γ, as well as lipotropic agents—choline, methionine, lipocaine, linethol, and linseed and maize oil. Other medicinal preparations prescribed are those of iodine, which reduce viscosity of the blood. Iodine may be taken during a period of 6 weeks followed by a 2 months' interval.

In the treatment of atherosclerosis very important is strict adherence to a definite routine in sleep and rest, annual leaves, as well as healthful occupational surroundings and living conditions.

Sanatorial and health resort treatment should preferably be undertaken at general-type sanatoria and in case of 1st-degree circulatory insufficiency, at cardiologic health centres.

HYPERTENSIVE VASCULAR DISEASE (MORBUS HYPERTONICUS)

Hypertensive vascular disease is a systemic malady. Its cardinal manifestation is elevation of both the systolic and the diastolic arterial pressure. This disease should be differentiated from symptomatic hypertension, which is only one of the symptoms of such diseases as nephritis, atherosclerosis, tumours of the pituitary body and the adrenal glands, poliomyelitis, encephalitis, etc.

Aetiology and pathogenesis. Different views of the aetiology of hypertensive vascular disease are current. The best-grounded of them is the neurogenous corticovisceral theory which was advanced by G. Lang and is based on I. Pavlov's physiologic teachings. According to this theory, in hypertensive vascular disease there is neurosis of the vasomotor centre, which results in a deranged regulation of the vascular tone. Clinical observations show that hypertensive vascular disease comes on in the wake of prolonged and severe emotional and nervous unrest. This results at first in temporary and then permanent tonic contractions of smaller arterioles and in elevation of the diastolic pressure. As a result of this, the systolic pressure also grows because the left ventricle has to work at greater load in order to overcome the increased resistance in the arterial system.

Prolonged spasm of the arterioles is in its turn responsible for the development of sclerotic changes in the smaller vessels, which contribute to the onset of persistent hypertension and its subsequent progression.

An important role in the development of hypertensive vascular disease is also played by the kidneys. The normal kidney is known to produce a protein substance—renin, whose secretion into the blood becomes particularly intense when the latter's supply to the kidneys is inadequate. Renin by itself does not produce hypertension but, when in combination with α_2-globulin, hypertensinogen secreted by the liver is converted into hypertensin, an active substance causing an elevation of the arterial pressure. In view of the discovery of the pressor properties of hypertensin, many authors, particularly in the United States, are inclined to regard a circulatory disturbance in the kidneys as the principal cause of hypertensive vascular disease. It has been proved by observations of Soviet authors, however, that at earlier stages of hypertensive vascular disease no changes occur in the kidneys. Moreover, even in persons who have died of this disease changes in the kidneys are not always found at autopsy. Consequently, the renal factor cannot be considered an early and principal cause of hypertensive vascular disease. At the same time, in the presence of hypertensive vascular disease the renal factor contributes to a further increase in the arterial pressure. Quite a few suppositions have been expressed concerning the leading role of the endocrine system in the development of hypertensive vascular disease. It is known that an increase in the function of the pituitary body leads to an elevation of the arterial pressure since the hormone of its posterior lobe—vasopressin—produces a hypertensive effect. In addition, the adrenocorticotropic hormone of the pituitary body may elevate the arterial pressure by acting on the adrenal glands and boosting their output of epinephrine and corticosteroids (cortisone, mineralocorticosteroids, etc.), which also cause hypertension. The latter may also be due to a hyperfunction of the cortical and medullary layers of the adrenal glands and an increased secretion of vasopressor hormones.

The question of connection between the genital glands and the arterial pressure has long been a subject for debate. It is known that an impairment of function of the genital glands (menopause, castration, etc.) leads to hypertension, while in vascular spasm the sexual hormones produce a vasodilating effect. That is why they are used for the therapy of a hypertensive condition. It has been found by now, however, that in a decreased function of the genital glands hypertension is caused by a functional disturbance in the nerve centres regulating the blood pressure rather than by that in the genital glands. Hormonal disturbances expose or aggravate the pathologic process taking place in the brain cortex. In toxic dystrophy of the thyroid gland hypertension is encountered also; it is not identical with hypertensive vascular disease, however, since only the systolic pressure is elevated while the diastolic pressure remains normal or is even reduced. The above-listed factors make it obvious that endocrine disorders play a subsidiary rather than principal role in the development of hypertension.

Pathologic anatomy. In the initial period of hypertensive vascular disease no morphologic changes occur in the body. Later, the intima of the vessels, particularly of the coronary and cerebral vessels, is infiltrated by lipoids and atherosclerotic plaques. A similar picture is also observed in the arteries of the kidneys, the spleen and the pancreas. Arteriolosclerosis develops in the arterioles. Necrotic lesions are sometimes found on their walls. Arteriolosclerosis may be circumscribed (selective) or diffuse in character. In arteriolosclerosis of the brain, spasms occur in the cerebral vessels, and the brain substance itself suffers damage, as is manifested by the emergence of isolated foci of brain softening and a disturbance in the cerebral circulation. In the initial stages of the disease, the kidneys are not involved. Later, hyalinosis and arteriolosclerosis developing in the kidney arterioles cause cicatricial contraction of the glomeruli, so that the latter diminish in volume (primary contraction of the kidney—nephroangiosclerosis). At the same time, scanty haemorrhages and degenerative changes are found in the tubules. Long-standing hypertension causes hypertrophy of the left cardiac ventricle and atherosclerosis of the coronary vessels later joined by cardiosclerosis.

Clinical picture. Hypertensive vascular disease is prevalent among middle-aged and elderly persons and sometimes occurs at a young age. In women, it most commonly appears for the first time, or is aggravated, in the climacteric period. This disease is known to have a number of developmental stages: initially, it is an exclusively functional disorder, later it causes organic lesions in vessels, and still later symptoms of insufficiency of sclerosed organs come on the stage. Naturally, both the patient's complaints and objective findings vary with the stages of the disease.

In the initial period the patient complains of intermittent headache, dizziness, tinnitus, insomnia, early fatigue and debility, pulsation of vessels in the temples and neck, a feeling of heaviness in the head, flushes, sometimes numbness of the fingers, and the creeps. The arterial pressure is elevated only in emotional unrest, physical exertion, and fatigue. After elimination of the causes of functional changes the patient is relieved of all symptoms and the arterial pressure returns to normal. Later, with the development of organic changes in the cardiovascular system, the rise in the arterial pressure becomes persistent although it varies insignificantly within the limits of 10-30 mm Hg, depending on rest and physical exertion, emotion, a change in the barometric pressure, etc. In this phase headache, dizziness and other symptoms become permanent. At the same time, there is enlargement of the left cardiac ventricle, the second sound over the aorta is accentuated, while the apex beat becomes elevating, the pulse, tense, and the peripheral arteries, tortuous. In the subsequent course of hypertensive vascular disease symptoms of insufficiency appear in individual organs worst affected by atherosclerosis. It should be noted that not all organs are involved uniformly. In the majority of cases, the disease affects the heart, in one of every three cases changes in the brain predominate, and in one of every ten cases there is arteriolosclerosis of the kidneys. The most frequent, however, are mixed forms with a simultaneous affection of the brain, heart and kidneys. Patients with a prevailing affection of the heart complain of periodic pains in the heart due to spasm of the coronary vessels and ischaemia of the heart muscle, breathlessness, a feeling of suffocation due to a severe left ventricular failure, which commonly occurs at night (cardiac asthma). In some cases, the patient dies of myocardial infarction or cardiovascular failure. In the case of prevailing affection of the brain vessels there is periodic spasm of the latter, which is manifested by sudden headaches, vomiting, fainting, and deafness. The most severe complication is brain apoplexy with pareses or paralyses of the extremities secondary to a prolonged spasm, thrombosis, or rupture of vessels, resulting in haemorrhages in the surrounding tissue, which later becomes softened. Cerebral haemorrhage is preceded by intense headaches, dizziness, speech disturbance (dysarthria), sometimes convulsions, etc. In apoplexy, the patient is unconscious, with a hyperaemic face, and deep and loud breathing. The state of unconsciousness may last as long as a few days. Cerebral haemorrhage is followed by paralyses of the extremities and a disturbance in the function of the pelvic organs. In thrombosis of the brain vessels, the disease runs a slower course.

It is necessary to dwell in detail on hypertensive crisis, which is accompanied by a steep rise in the arterial pressure and neurovascular disturbances. Hypertensive crisis is, as a rule, due to neuro-emotional upset, and sometimes meteorologic factors, changes in the atmospheric pressure. Crises are encountered both in the initial and subsequent stages of hypertensive vascular disease. Hypertensive crises are usually subdivided into two types. The first type, observed in the early stage of hypertensive disease, is of brief duration (lasting from several minutes to 2-4 hours), and is manifested by general nervousness, extreme excitation, sweats, intense headaches, dizziness, tinnitus, trembling of the extremities, breathlessness, and sometimes by the appearance of a "veil" or mist before the eyes. There is a supposition that crises of the first type are caused by a brief secretion of excess ephedrine. Crises of the second type, or late crises, are manifested by headaches, debility, nausea, vomiting, a drastic impairment of vision due to spasm of the retinal vessels and oedema in the surrounding tissues. There is also deterioration of hearing, asthma and cardiac pains. The arterial pressure is elevated and the pulse is tense. Late crises are due to a prolonged secretion of excess norepinephrine (arterenol), as a result of which they run a longer course. Changes in the kidneys are very characteristic. The analysis shows albuminuria and haematuria, the concentrating power of the kidneys is decreased, the nonprotein nitrogen level in the blood is heightened and renal insufficiency develops in the further course of the disease process (polyuria, stable low specific gravity, etc.) and azotaemic uraemia is observed. There is growing anaemia, which is especially pronounced in rapidly progressing forms of hypertensive vascular disease. In the late stages of hypertensive disease visual disorders make their appearance. In the early period of the disease the retinal vessels are unchanged or there is constriction and tortuousness of the arteries. Later, the initial signs of sclerosis of the retinal arteries appear. The arterial walls become thickened, and at the point of intersection of the arteries with veins the latter are compressed. This symptom described by Gunn and Salus is manifest in a variable degree, depending on deformation of the veins and their compression into the retina. One may also note haemorrhages, oedema of the optic papilla and choked disk. The retinal tissue shows white, sometimes yellow foci, at times seen as a star around the macula lutea (yellow spot of the retina). The cause of these foci is attributed to a severe trophic disturbance in the retina. The intestinal symptoms are flatulence, a feeling of fulness in the epigastrium, with a simultaneous feeling of compression in the heart region. The latter is due to a high position of the diaphragm and a reflex disturbance in the blood flow in the coronary arteries.

The peripheral blood is found to have erythrocytosis and, in arteriolosclerosis of the kidneys, anaemia.

Electrocardiographic changes in hypertensive vascular disease vary with the stage of the disease process. In the initial stages the electrocardiogram is normal. Later, because of left ventricular hypertrophy there is a left axis deviation (sinistrogram), sometimes a depressed *S-T* segment and changes in the *T* wave. These changes may be secondary to cardiac insufficiency and myocardial dystrophy of the left ventricle. Electrocardiographic changes in coronary insufficiency (angina pectoris) or myocardial infarction become manifest (see "Myocardial Infarction"). X-rays show dilatation and elongation of the aorta and left ventricular hypertrophy (aortal heart).

The biochemical structure of the blood also undergoes a change. In the initial period of the disease the amount of cholesterol and its fractions is within normal limits and subject to insignificant variations, whereas in far-advanced atherosclerosis the cholesterol level is elevated and certain changes occur in the protein fractions of the blood.

With regard to its course, hypertensive vascular disease is subdivided into two forms: (a) slowly progressing form; and (b) rapidly progressing form. The former may in its turn be subdivided into three stages. The first stage is characterized by a drop in the arterial pressure to normal, transient spastic contractions of vessels, though unaccompanied by sclerotic changes. The second stage is characterized by vascular sclerosis, with unmarked changes in the heart, kidneys and retina. The arterial pressure falls, though it is still short of the normal level. In the third stage, the arterial pressure is high and fails to decline in response to therapy. Sclerotic changes in the heart, kidneys and brain are more severe than in the second stage: there is a considerable functional disturbance in these organs, which is manifested by a third-degree circulatory insufficiency, azotaemia, the appearance of circulatory disorders in the brain, with pareses and paralyses, and haemorrhages in the retina. The slowly progressing form of hypertensive disease is subdivided into the cardiac, cerebral, renal, and mixed forms (variants), according to the prevailing involvement of a particular organ.

The rapidly progressing form runs a malignant course lasting not longer than 1-2 years, is accompanied by affection of the kidneys, anaemia, and ends in azotaemic uraemia as a result of malignant arteriolosclerosis of the kidneys. The clinical picture is characterized by symptoms of hypertensive vascular disease concurrent with involvement of the kidneys. The modern methods of therapy sometimes help transform this form into a slowly progressing form of hypertensive disease.

Differential diagnosis. It has been pointed out above that not any rise in the arterial pressure is a sign of hypertensive vascular disease. There are quite a few diseases which run their course with symptomatic hypertension, for example, nephritis, constriction of one or both renal arteries, organic lesions of the central nervous system (encephalitis, tumours, poliomyelitis, closed cranial injuries), as well as lesions of larger vessels and the aorta, and heart defects (stenosis of the left atrioventricular orifice, constriction of the aortic orifice, congestive symptoms). Elevation of the arterial pressure in heart defects with a circulatory disturbance, as well as in pulmonary fibrosis and pulmonary emphysema, is most probably caused by hypoxia and anoxia of the central nervous system, whose stimulation causes spasm of the arterioles and a rise in the arterial pressure. An increase in pressure is also produced by some endocrine disorders. Among them are Itsenko-Cushing's disease, phaeochromocytoma, etc. In Itsenko-Cushing's disease the pituitary body and adrenal glands are affected. The principal symptoms are as follows: obesity with comparatively lean and thin extremities, hairiness (hirsutism), a reduced sexual interest, impotence in men, a disturbance in the menstrual cycle in women, erythraemia, hyperglycaemia, osteoporosis, predisposition to bleeding in the absence of thrombopenia, a moon-shaped puffy face, and purple-blue tension lines (striae) on the stomach wall.

A symptomatic increase in the arterial pressure is seen with tumours of the cerebral layer of the adrenals (phaeochromocytoma) in connection with an increased secretion of epinephrine and arterenol. In phaeochromocytoma the arterial pressure is for the most part elevated periodically, in paroxysmal attacks, although it may rise gradually and remain at a high level for a long time. It is identified from the presence of epinephrine in the blood during an attack, by x-rays (pneumoretroperitoneum-pneumoren). Upon excision of the tumour the arterial pressure returns to normal and all other symptoms disappear without leaving a trace.

Nephroangiosclerosis has to be differentiated from a secondary contraction of the kidney due to chronic nephritis. In certain cases, differential diagnosis is extremely difficult. Evidence of the presence of nephroangiosclerosis is high-degree hypertension, marked hypertrophy of the heart muscle and signs of growing uraemia with relatively slight changes in the urine: an insignificant amount of protein and a low red cell count. A careful consideration of the patient's complaints is very important for a differential diagnosis. Chronic nephritis is recognized by symptoms pointing to a past history of acute nephritis (blood-stained urine, puffiness under the eyes, headaches combined with breathlessness, etc.).

Prognosis. Prognosis depends on the form and stage of the disease. A disease without complications and crises sometimes lasts for years; the patient, however, is always exposed to the danger of cerebral haemorrhage and spasm or thrombosis of the coronary vessels culminating in myocardial infarction. Patients with a rapidly progressing form of the disease soon become debilitated. Patients with a slowly progressing form retain their working capacity if no complications occur in the heart, kidneys and brain and on condition that their work, leisure and diet have been adequately rationalized. In rehabilitation special measures should be taken to eliminate the neurogenous factors damaging the patient's mental health.

Prophylaxis and treatment. Prophylaxis consists in measures to ensure healthful working and living conditions. In the Soviet Union, the absence of unemployment, considerable improvement of the housing and living standards, advancement of physical culture and sports on a nation-wide scale, availability of health resorts and holiday homes to the entire population, and elimination of occupational health hazards in industry and agriculture have provided favourable conditions for the control of hypertensive vascular disease. Prophylactic measures consist in regular examination of the arterial pressure among the population, especially in persons above 30 years of age. Persons found to have the initial form of hypertensive vascular disease are kept under observation by medical institutions.

The treatment of hypertensive vascular disease consists in measures to restore the normal cortical regulation of the arterial pressure by means of a hygienic way of life and drug therapy. The patient must be enjoined to have sufficient rest and sleep. His neuropsychic sphere is protected against disturbing emotions by eliminating all factors likely to damage his mental health as well as by administration of bromides, tincture of valerian, etc. In the initial stages of hypertensive disease it is possible to reduce the arterial pressure by administrations of dibazol, luminal, papaverine with theophylline (see formula No. 21), intramuscular or intravenous injections of 10 ml of a 25 per cent magnesium sulphate solution (formula No. 87) as well as serpasil or reserpine, which are given in a dose of 0.1-0.2 mg three times daily during a period of 10-25 days. The so-called maintenance doses of 0.1 mg may also be given for a long time. Raunatin (ghendon) is prescribed in a daily dose of 2 mg to be taken after meals during a period of 10-15 days. Dibazol is taken per os in a dose of 0.02-0.05 g three times daily or is introduced intramuscularly in the form of a 1 per cent solution to be administered in a 2 ml dose daily during a period of 5-10 days. Ganglioblocking agents may also be prescribed, such as pentamine 0.02 g to be given 2-3 times daily, or hexonium (hexamethonium) per os, 0.1 g three times daily or intramuscularly in a dose of 1 ml of a 2 per cent solution. Ganglioblocking agents block the synapses of the ganglia of the vegetative nervous system and stop the excessive flow of stimuli from the vegetative centres to the peripheral vessels. Recommended also are carbocholine (doryl) 0.01-0.02 g to be given 2-3 times daily during a period of 10-15 days), angiotrophin (padutin) subcutaneously in a dose of 1 ml, a 0.5 per cent progesterone solution injected intramuscularly in a 24-hour dose of 2 ml during a period of 10 days, and intramuscular folliculin, 5,000-10,000 mg in 24 hours for a period of 10-15 days. Cardiac insufficiency should be treated in accordance with the general rules. Headaches are allayed by mustard plasters applied to the gastrocnemius muscles, and to the occipital region, by the application of 6-8 leeches to the region of the mastoid processes, and administration of hot foot baths. The remedies indicated for a hypertensive crisis are confinement to bed, application of leeches, blood-letting, intravenous infusion of dibazol (1-2 ml of a 2 per cent solution twice daily), and intravenous infusion of a 10 ml dose of a 25 per cent magnesium sulphate solution.

The intake of food rich in cholesterol (eggs, fatty meat, brains, butter) is restricted, meat soups, gravies, piquant and salty dishes likely to produce thirst are forbidden. Common salt and liquids must be restricted. The patient must be given food rich in vitamins and totally abstain from tobacco and liquors.

ANGINA PECTORIS

Angina pectoris, otherwise known as stenocardia, is prevalent among persons of advanced and old age, and is manifested by paroxysmal pains in the heart or retrosternal pains, which radiate to the left arm, shoulder-blade, neck, etc. Pains persist from several minutes to half-an-hour, and sometimes longer.

Aetiology and pathogenesis. The causes of angina pectoris are quite variable; in the majority of cases, however, anginose pains are due to organic and, to a lesser extent, functional changes in the coronary arteries of the heart. The major cause of organic lesions in the coronaries is most commonly atherosclerosis, less often rheumatic coronaritis or syphilitic constriction of the orifices of the coronary arteries. In certain cases, angina pectoris is found in the absence of atheromatosis of the coronary vessels, i.e., is exclusively functional in origin. This is due to the influence of an intense nervous and emotional upset, or reflex stimuli from the organs of the abdominal cavity (stomach, gallbladder, etc.) if the latter undergo pathologic changes. In such cases the nervous regulation of the coronary circulatoin is usually deranged. The appearance of pains is attributed to a disturbance in the coronary blood flow, a diminished blood supply of the heart muscle and ischemia (hypoxia) of the latter, as well as accumulation in the heart muscle of incompletely oxidized metabolic products which cause stimulation of the intracardiac nerves. Through the extracardiac sympathetic and parasympathetic nerve pathways and via the sympathetic nerve ganglia these stimuli are conveyed to the corresponding segments of the spinal cord and thence to the medulla oblongata and the cerebral cortex. From the cerebral cortex the stimuli are transmitted to the sensory endings of the peripheral nerves ramifying in the skin of the chest in the region of the heart, and in the skin of the left shoulder, the left arm and neck. In addition, various stimuli from the skin or the visceral organs (liver, stomach, intestine, etc.) may be transmitted to the cerebral cortex through these nerve pathways. Stimuli, acting in the reverse direction on the coronaries, may produce their spasm and an attack of angina pectoris. In some cases, an attack coincides with an additional stimulation (for example, the drone of an airplane), which results in the formation of a conditioned reflex association. Later, cardiac pains appear even after an additional stimulation of the cerebral cortex. Such attacks of angina pectoris are known as conditioned reflex paroxysms.

Pathologic anatomy. In the majority of cases, there is atherosclerosis of the coronary arteries. The latter are narrowed, and a simultaneous sclerosis of the heart muscle develops. Sometimes, the coronary arteries are unchanged; however, one can note an increased permeability of the vessel wall, swelling of the intima, and the presence of effusion in the lumina of the vessels, apparently caused by myocardial ischemia.

Clinical picture. The patient complains of a feeling of oppression in the chest, pains of variable intensity in the chest and behind the sternum, which may be compressing, pressing, stabbing, and lancinating in character and radiate to the left shoulder and down the arm to the elbow and fingers, to the lower jaw, sometimes to the right shoulder, the right shoulder-blade, and the back of the neck. The patient is rigid because pains grow worse on moving; the face is pale, covered with cold perspiration, and its expression shows fear and suffering. Pains usually come on suddenly, sometimes at rest (angina at rest) or after a hearty meal, emotional unrest, or physical exertion (angina of effort). Stenocardia at rest is associated with considerable atherosclerotic changes in the coronary arteries. It sets in at night during sleep or when a person is in a recumbent position because of an intensified vasoconstrictive action of the vagus nerve in sleep and at rest. Examination of Zakharyin-Head's zones in the region of the left arm, neck, sternum and back reveals areas of hypersensitive skin from the 7th cervical vertebra to the 5th upper thoracic vertebra. The heart shows no substantial abnormality. The electrocardiogram exhibits a disturbance in the blood flow, the *S-T* segment is depressed, while the *T* wave may be inverted. Soon after the cessation of an attack, however, the electrocardiographic picture returns to normal. During an attack no elevation of body temperature or changes in the peripheral blood are, as a rule, noted. Sometimes, during an especially severe paroxysm of pain the body temperature may rise insignificantly, with an accompanying slight leucocytosis, which comes to an end soon after the cessation of the attack.

Differential diagnosis. Identification of angina pectoris is not difficult. It should be only borne in mind that pains may be felt in unusual places—in the epigastrium or in the right hypochondrium, which may give cause for an erroneous diagnosis of peptic ulcer or cholecystitis. Pains similar to those in angina pectoris may be felt in pleurisy, intercostal neuralgia, myositis, and injuries to the ribs and costal cartilages, which are identified by means of conventional examination. Syphilitic arthritis is diagnosed by considering the patient's complaints, x-rays, and Wassermann's test. Angina-like attacks are seen in patients with diaphragmatic hernia and an ulcer localized in the cardial portion of the stomach when stimulation of the vagus nerve gives rise to a reflex spasm of the coronary arteries with pains in the precordium. The latter may occur also in subjects with sclerosis of the aorta, syphilitic aortitis, and rheumatic coronaritis. Sclerotic changes in the aorta narrow the lumina of the coronary arteries branching off from it and thereby impair the conditions of blood flow to the heart muscle. Possibly, pains are also caused by stimulation of the nerve endings of the outer coat of the aorta. They fail to subside after the administration of nitroglycerin. Anginose pains may be produced by a trophic disorder in the heart muscle secondary to aortic valve insufficiency, stenosis of the aortic orifice, etc. The same pains also occur in acute anaemic conditions, chronic nephritis, and left ventricular hypertrophy when the oxygen supply of the heart muscle is inadequate. Sometimes, precordial pains are due to neurosis. They are felt around the nipple and may be nagging or stabbing, lasting for hours and days and failing to respond to nitroglycerin. Such pains occur in combination with other manifestations of neurosis: headaches, tearfulness, etc. It is highly important to establish a differential diagnosis between angina pectoris and myocardial infarction, since a belated identification of myocardial infarction may have regrettable consequences.

Prognosis depends on the principal disease. The outlook is brighter in angina of effort. During the latter, attacks are at first rare but later become more frequent, recurring almost every day. Angina at rest runs a more severe course because its cardinal cause is atherosclerosis of the coronary arteries, which tends to aggravate and may result in myocardial infarction. The prognosis of angina pectoris, both of effort and at rest, is more favourable upon elimination of physical and mental overstrain. The working capacity of subjects suffering from frequent anginal attacks is considerably reduced. They may be employed on jobs unlikely to involve fatigue, physical exertion and neuro-emotional unrest.

Prophylaxis of angina pectoris consists in measures to eliminate its precipitating causes, therapy of atherosclerosis, rheumatic fever, syphilis, diseases of the gastrointestinal tract (cholecystitis, peptic ulcer, colitis, etc.), and neuropsychic and angioneurotic processes responsible for spasm of the coronary arteries. In measures to control angina pectoris emphasis should be laid on the inculcation of hygienic habits since childhood. Physical culture and sports, hardening of the body, abstinence from smoking, alcohol, and overeating, as well as measures to ensure a normal quiet situation both at work and at home largely contribute to the prevention and treatment of angina pectoris.

Treatment. In the case of an anginal attack one should immediately take nitroglycerin, which produces dilatation of the coronary arteries and terminates the attack in a matter of 2-3, less often 5 minutes. Nitroglycerin in drops or in tablet form is absorbed by the oral mucosa, therefore it is taken in a dose of 2-3 drops put on a piece of sugar, which is placed on or under the tongue. Nitroglycerin may be given in tablets of 0.0005 g (formula No 15*a*). Another remedy is amyl nitrite in ampules, which are wrapped in a handkerchief and crushed so as to inhale its vapour. Very helpful to some of patients is validol, which is given in a dose of 5 drops on a piece of sugar placed under the tongue, as well as its substitute, Zelenin's drops. In very severe anginal attacks it is necessary to give the sufferer an hypodermic injection of a 1 ml dose of a 1-2 per cent morphine or omnopon solution in combination with 0.5 ml of a 0.1 per cent atropine sulphate solution. In mild cases, an anginal attack may be aborted by the application of hot baths for the feet and hands, or mustard plasters to the heart region. After a prolonged anginal attack the patient should be confined to bed for several days.

The treatment of patients in the period of remission consists in peroral administrations of papaverine with luminal and euphyllin (formula No. 24), dibazol, nitranol* (formula No. 16), nitropenton * (formula No. 16*a*), tiphen (formula No. 16*b*), and khellin in a dose of 0.02 g 2-3 times daily (formula No. 196). Iodine preparations are also prescribed, especially for atherosclerosis. Angina pectoris has of late been treated with ganglerone in powder form in a dose of 0.04 g 3 times daily, or in the form of a 1.5 per cent solution given in one teaspoonful dose 3 times daily or intramuscularly in a dose of 2 ml 2-3 times daily. Of the physiotherapeutic procedures, total d'arsonvalization, diathermy and ionogalvanization, irradiation of the cervical sympathetic ganglia with the light of a mercury-quartz lamp in erythema doses, and saline-pine baths are used.

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* Nitranol and nitropenton are contraindicated for glaucoma.

Persisting angina pectoris requires surgical interference: trans-sternal blocking of the cardiac and aortic plexuses, and intrasternal drip anaesthesia with 100 ml of a 0.5 per cent novocain solution introduced into the sternal marrow. Unfortunately, all of the above-mentioned methods, just as bilateral ligation of the internal thoracic artery so as to improve the blood supply of the heart muscle, produce only a brief amelioration in the patient's condition.

MYOCARDIAL INFARCTION (INFARCTUS MYOCARDII)

Focal necrosis of the heart muscle due to a prolonged ischemia secondary to diminished coronary flow or dystrophic changes in the heart muscle is known as myocardial infarction.

Aetiology and pathogenesis. Myocardial infarction commonly affects men of 40 to 60 years of age, less often younger persons. The incidence of this disease among men is 3-4 times that among women. The most frequent cause of myocardial infarction is atherosclerosis of the coronary arteries, which leads to their mechanical constriction and, in addition, impairs the reactivity of the vascular walls. As a result of this, during intense exertion the coronary arteries become stenosed rather than dilated. Myocardial infarction also results from hypertensive vascular disease and a great emotional stress, which is evidence of the cardinal role played by the cerebral cortex in a disturbance of the coronary blood flow.

The development of myocardial infarction is also attributed to neuroreflex influences emanating from the stomach, liver, intestine, etc. Myocardial infarction often comes on at night during sleep when the coronary flow is decelerated due to an increased vagus nerve tone. Cases are known, infrequent as they are, of myocardial infarction developing as a result of coronary embolism in subacute bacterial endocarditis, rheumocarditis, and syphilitic affection of the aorta, as well as due to a prolonged spasm of the coronary arteries in which no atheromatosis is found.

Pathologic anatomy. Myocardial infarction commonly develops in the anterior (anterior infarction), posterior (posterior infarction) and in the lateral wall of the left ventricle, and in the interventricular septum. This localization of infarction is due to the fact that a circulatory failure mainly occurs at the ramification of the left coronary artery, in particular in its descending and bypassing branches, which supply the anterior, posterior, and lateral walls of the left ventricle and the interventricular septum. Occlusion of the branches of the right coronary artery gives rise to infarction of the posterior wall of the right ventricle and the posterior part of the interventricular septum, in connection with which various disturbances occur in the conduction system of the heart. In myocardial infarction the left ventricle is damaged more often and more severely than the right ventricle, since the latter has a thinner musculature and is supplied with blood from the thebesian veins communicating with the right ventricle and through anastomoses from the left coronary artery. No isolated lesions of the interventricular septum are known to occur. They are usually found simultaneously with anterior or posterior infarction. Simultaneous infarction of the lateral and anterior walls is known as anterolateral infarction. Correspondingly, the term posterolateral infarction is used for a simultaneous necrosis of the lateral and posterior walls of the left ventricle.

During an increased work of the heart due to physical overexertion the ventricular wall may bulge and form an acute cardiac aneurysm. The latter may cause a rupture of the heart and its tamponade, i.e., cessation of the heart's action due to effusion of the blood into the pericardium. Myocardial infarction may be accompanied by pericarditis if the disease process extends toward the outer surface of the heart. In cases where necrosis reaches as far as the internal membrane of the heart there develops thromboendocarditis which may become a source of emboli entering various peripheral vessels of the brain, extremities and visceral organs, possibly with a fatal outcome.

Granulation connective tissue growth, i.e., cicatrization, begins to develop in the area of necrosis and softening. This process ends within 6 to 8 weeks, leaving a scar at the site of a former infarct. The healed part of the heart muscle, when large enough in diameter, may bulge under pressure from the blood, with formation of a chronic aneurysm. In a considerable strain likely to increase the intracardiac pressure, a rupture of the aneurysm with tamponade of the heart may ensue.

A person may have sustained several infarctions at different times in the past or simultaneously.

Clinical picture. The clinical picture of myocardial infarction was for the first time described by V. Obraztsow and N. Strazhesko (1909). The cardinal features of myocardial infarction are pain and acute failure of the coronary blood flow. The pain is sudden in onset, sometimes with loss of consciousness. It is usually substernal, being felt in the upper or lower region of the sternum (status anginosus), or may be located in the right hypochondrium or the epigastrium (status gastralgicus). At the same time, dyspeptic symptoms are also present, the abdomen is inflated, tender and tense. Pains radiate to the left arm, shoulder, neck, and fail to subside in response to nitroglycerin. In rare cases, myocardial infarction begins with an attack of cardiac asthma which forces the patient to assume a sitting or half-sitting posture (status asthmaticus); sometimes it is accompanied with manifestations of pulmonary oedema. The asthmatic form of myocardial infarction running its course with a clinical picture of left ventricular failure is characterized by a high mortality rate.

Myocardial infarction may be accompanied by symptoms of cardiovascular insufficiency. An especially formidable sign is vascular failure—a collapse with a fall of the arterial and venous pressure, a reduction in the velocity of the blood flow and in the mass of circulating blood. The patient's body is moist with cold and sticky perspiration, the face is ashen, and the pulse is frequent and thready, sometimes arrhythmic (extrasystolic paroxysmal tachycardia, atrial fibrillation, heart block). The appearance of an alternating pulse is a very ominous sign.

On auscultation of the heart, dull sounds are audible, and sometimes a pericardial friction rub, if the infarction is localized in the anterior wall or extends toward the outer membrane of the heart; a gallop rhythm may also be heard. Other developing symptoms are mainly those of left ventricular failure with pulmonary oedema, a considerable number of moist rales in the lungs and dyspnoea. In certain cases, the right ventricle is also weakened, with an enlarged and tender liver, swollen veins of the neck, and cyanosis.

Other important signs of myocardial infarction are a rise in body temperature and changes in the peripheral blood. Resorption of decomposition products in the area of necrosis is accompanied by fever. Usually, fever is present since the second day after the onset of infarction, reaching levels of 38 to 38.5°C. It should be kept in mind that in collapse the temperature remains normal or below normal. In the case of necrosis extending to the internal layers of the heart muscle, as far as the endocardium, a subfebrile temperature may persist for weeks in connection with parietal thromboendocarditis. The rise in temperature is attended by neutrophylic leucocytosis with a shift to the left in the blood count, and preponderance of rod and juvenile neutrophils, an elevated erythrocyte sedimentation rate, and a positive reaction to C-reactive protein. Leucocytosis persists for a brief period, seldom longer than a fortnight, and gradually declines to normal. The highest sedimentation rate within a range of 20-40 mm per hour is observed in the period of leucocytosis decline, and for this reason the curves of leucocytosis and the sedimentation rate cross. The degree of temperature rise and an increase in the white cell count depend on the size of the necrotized portion of the heart muscle and on body reactivity.

The most important findings for an early diagnosis of myocardial infarction, aside from alterations in the electrocardiogram, may also be certain enzymatic derangements in the body, in particular an increase in aldolase of the blood serum (above 12 units) and in transaminase (above 40 units), which are produced in excess in the necrotized portions of the heart muscle. In addition, an increased fibrinogen content of the blood (above 0.5 g) is also a significant symptom.

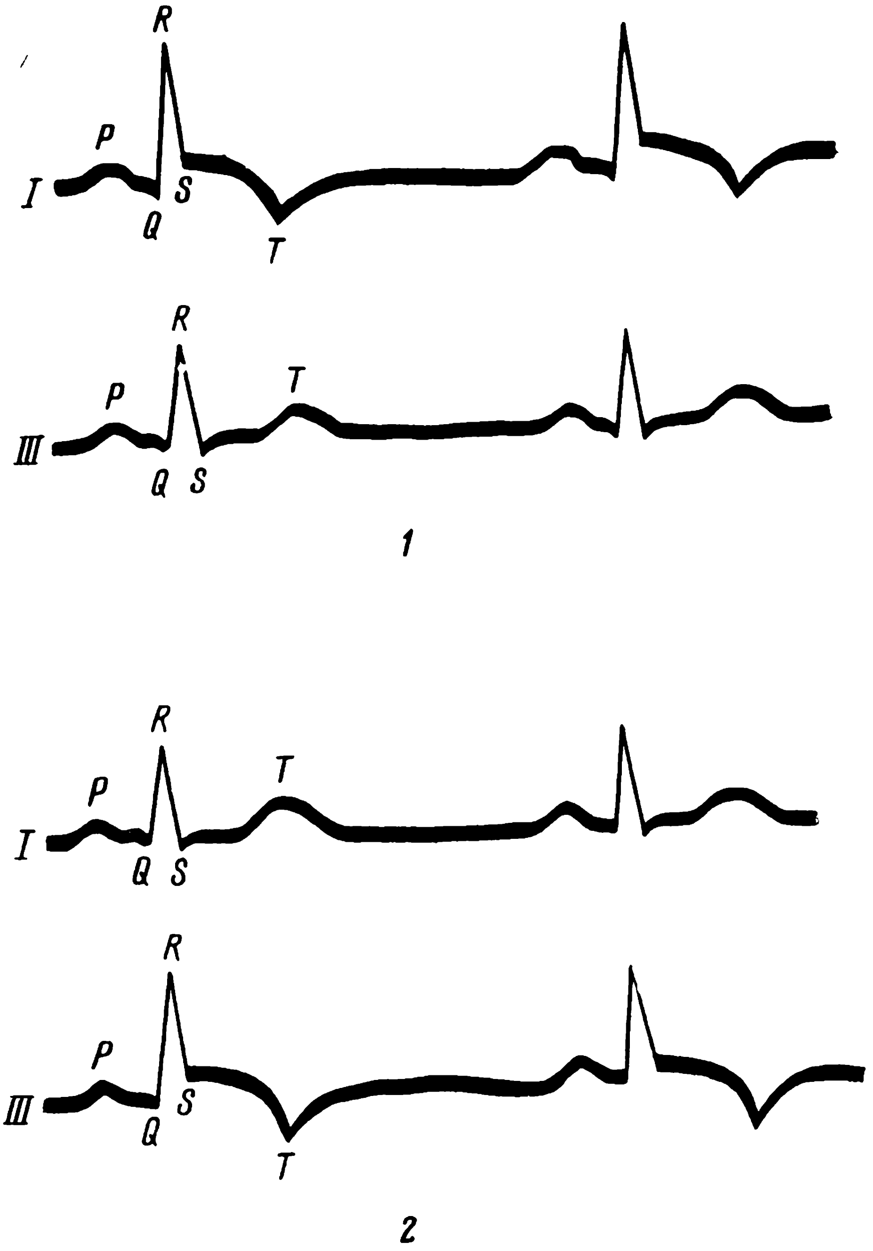

Fig. 44. Electrocardiogram in myocardial infarction
1—anterior wall; 2—posterior wall

An important role in identifying myocardial infarction and determining its site, depth, and extent of necrosis of the heart muscle is played by electrocardiographic examination (Fig. 44). Sometimes, electrocardiographic alterations characteristic of a fresh infarct may be noted as early as a few hours after an attack of pain due to myocardial infarction. When no alterations are discovered, as may be the case in the early hours of myocardial infarction, another electrocardiogram is required. Later the disease process in the heart muscle is studied by means of clinical and repeated electrocardiographic observation.

In infarction of the anterior wall of the left ventricle characteristic alterations are noted in precordial leads I and IV. In lead I the *T* wave is at first absent and later becomes negative, both in precordial leads I and IV. In lead III the *S-T* segment is depressed, in infarction of the posterior wall of the left ventricle an *RS-T* elevation occurs in lead III and depression in lead I. When the interventricular septum is involved in the disease process the signs characteristic of anterior or posterior infarction are concurrent with deflections in precordial leads II and III also. A lesion of the lateral wall of the left ventricle gives rise to typical alterations in precordial leads V and VI simultaneously with alterations characteristic of infarction of the anterior or posterior wall. In this way, detection of clinical and electrocardiographic abnormalities offers full opportunity for a timely diagnosis of myocardial infarction, which is highly important for emergency therapy. It should be noted, however, that in rare cases myocardial infarction may fail to be reflected on the electrocardiogram. In such cases the diagnosis of myocardial infarction must be based on its clinical manifestations.

Atypical forms. Atypical forms of myocardial infarction—peripheral, cerebral, and arrhythmic—are fairly frequent. In peripheral forms, the pains are at first felt between the shoulder-blades, in the left arm, the left elbow, the upper thoracic portion of the spine, in the oesophagus, the teeth, and in the entire lower jaw. In such cases, in the absence of electrocardiographic evidence, myocardial infarction may be mistaken for myositis, periostitis, etc. The first symptoms of the cerebral form are intense headaches and syncope. There are often hemiplegia, hemiparesis, which are later followed by pains in the heart region. The cerebral form is characterized by a reflex spasm of the cerebral vessels and the passage of emboli from the left ventricle in thromboendocarditis. A characteristic feature of the arrhythmic forms is the appearance of paroxysmal arrythmia or cardiac fibrillation. These forms, just as the cerebral form, are the most dangerous to the patient's life.

Complications of myocardial infarction are variable. Among them is rupture of the heart, which is observed with an acute and chronic aneurysm of the left ventricle. Rupture of an acute aneurysm is encountered between the 2nd and 14th days of the disease and causes death within a matter of 2-3 minutes, less often within a few hours. An aneurysm may be considered in every case of acute infarction running its course with a prolonged pain and fever, leucocytosis, and an elevated erythrocyte sedimentation rate. In a patient with a cardiac aneurysm pulsation of the heart is visible and felt on palpation inwardly to or above the site of the apex beat. This is encountered in the 2nd or 3rd week of infarction. At the same time, the apex beat is displaced to the left and downwards. On auscultation of the heart dull sounds are heard, as well as diastolic and systolic murmurs over the site of the aneurysm. A chronic cardiac aneurysm gives rise to chronic circulatory insufficiency. Another complication of myocardial infarction is a rupture of the interventricular septum. In this case, there is a sudden appearance of a loud systolic murmur and a thrill over the base of the heart and the entire heart region.

Necrosis of a papillary muscle due to myocardial infarction produces mitral insufficiency with a systolic murmur at the heart apex differing from that heard upon rupture of the interventricular septum. Other serious complications are paroxysmal tachycardia and cardiac fibrillation. The latter sometimes appears without precursory symptoms, and is apparently associated with a total spasm of the coronary arteries. Pulmonary infarction and oedema should also be described as complications of myocardial infarction. The former is most commonly of embolic aetiology, and is encountered with parietal thrombi of the right cardiac ventricle. In certain cases pulmonary infarction is secondary to stasis in the pulmonary circulation and thrombosis of the pulmonary vessels caused by a left ventricular failure, the usual concomitant of myocardial infarction. The diagnosis of pulmonary infarction rests on the patient's characteristic complaints and objective findings. These complaints are chest pains, dyspnoea. palpitation, cough with a blood-stained sputum, fever of up to 38°C and higher, and sometimes pneumonia.

Differential diagnosis. Myocardial infarction should above all be differentiated from angina pectoris. In myocardial infarction pains are characteristically intense and prolonged. As distinct from anginal these pains fail to respond to nitroglycerin which gives only a slight soothing effect. A state of anxiety is characteristic of myocardial infarction, whereas in angina pectoris the patient may remain stiff for hours. As distinct from myocardial infarction, angina pectoris produces no collapse, while the pulse is full and rhythmic, with no cardiac insufficiency, no fever and no changes in the peripheral blood. The electrocardiogram shows no alterations characteristic of the initial period of myocardial infarction. The asthmatic forms of myocardial infarction must be differentiated from cardial asthma. The best method for this purpose is electrocardiography. Myocardial infarction may be confused with food poisoning, since the latter gives certain similar symptoms, such as nausea, vomiting, pain, fever and leucocytosis. The diagnosis, apart from the electrocardiogram, is based on a study of the patient's anamnesis pointing to a history of anginal attacks. Often, a perforating ulcer of the stomach or the duodenum, as well as other diseases producing a picture of acute abdomen, are mistaken for myocardial infarction, particularly that of the posterior wall.

The diagnosis of myocardial infarction is aided by information on the patient's age, the absence of symptoms of peptic ulcer, a disturbance in the cardiovascular system, as well as electrocardiography. In certain cases, myocardial infarction has to be differentiated from spontaneous pneumothorax, renal colic, and embolism of the pulmonary artery.

The *prognosis* of myocardial infarction is always serious, especially in persons with multiple diffuse and wide areas of damage and repeated infarctions. One ought to display great caution in the early days of infarction when even the slightest physical strain, effort in defecation, let alone early rising from bed, are contraindicated. A patient with microfocal infarctions may be considered practically healthy in 3-4 months, and one with macrofocal infarctions, in 6-7 months. Following an infarction the patient must abstain from physical and mental overexertion during 1-2 years. He may resume working only if his job needs no physical overstrain.

The *prophylaxis* of myocardial infarction consists in the treatment of atherosclerosis and measures to safeguard the patient against mental and physical strain, normalize his sleep and rest, and eliminate diseases of the abdominal organs, which may become the foci of reflex influences on the coronary arteries. Another prophylactic measure is the administration of anticoagulants if the prothrombin level is elevated. Also steps are sometimes taken to uncover a concealed disturbance in the coronary circulation by undertaking a functional test in the form of a dosed physical strain and electrocardiography before and after the test. Evidence of coronary insufficiency is depression of the *S-T* segment below the isoelectric line or inversion of the *S* wave in all or in some of the leads. An important role in the prevention of myocardial infarction is played by exercise therapy, sports, treatment at cardiologic sanatoria and abstinence from smoking and alcohol.

Treatment. Persons with myocardial infarction are subject to hospital treatment. They are brought to a hospital on a stretcher after a preliminary administration of analgesics and cardiovascular drugs. Special ambulances have of late been made available in large cities for transporting patients with myocardial infarction.

Pains are allayed by a hypodermic injection of 1-2 ml of a 1 per cent morphine solution together with 0.5 ml of a 0.1 per cent atropine solution; the latter is introduced in order to improve the patient's tolerance of morphine and reduce the vagus nerve reflex (formula No. 35). A marked weakening of the heart muscle, severe tachycardia, cardiac asthma, or a considerable drop in the arterial pressure call for a slow intravenous infusion of 1 ml of strophanthin or corglycon (formula No. 12) in combination with sympathol, mesaton, glucose, as well as cordiamine, caffeine and camphor injected subcutaneously. The state of collapse is sometimes relieved by 500 ml of a 5 per cent glucose solution, 1 ml of strophanthin, 1 ml of mesaton, 1 ml of noradrenaline, and 5,000 to 10,000 U of heparin introduced by intravenous drip. A patient recovered from a collapse is given spasmolytics (luminal, papaverine, euphyllin) (formula No. 24), and anticoagulants (neodicoumarin, 0.05 g; pelentan, 0.15 g; phenylene, 0.03 g 2-3 times daily). Sometimes, heparin is also administered during the first 2-3 days in a dose not larger than 30,000-50,000 U per treatment course.

Anticoagulants are administered with a check being kept on the prothrombin content of the blood, because they are likely to produce a profuse bleeding in consequence of a sharp reduction in the coagulation of the blood. It is desirable to bring the prothrombin level to 40 per cent. Treatment with long-acting anticoagulants (pelentan, neodicoumarin, etc.) is continued for 3-4 weeks. The prothrombin level must be determined at first once in every 2-4 days, later every five days.

In addition to examination of the blood prothrombin, urinalysis should be carried out every day and anticoagulant therapy should be discontinued upon the appearance of haematuria. In proneness to bleeding vitamin K (vicasol) is prescribed in a dose of 0.015 g to be taken three times daily. Anticoagulants are contraindicated for diseases of the kidneys, the liver, the bile ducts, gastric and duodenal ulcer, and haemorrhagic diathesis.

In myocardial infarction, a rational dietary pattern is very important for successful therapy. The patient is strictly confined to a prolonged bed rest, which in case of an incorrect, especially excessive diet may lead to an increment in body weight and pathologic symptoms in the alimentary organs, increased flatulence and constipation and to anginal attacks. Therefore the patient's diet is gradually widened in respect of calorific value, volume, manner of cooking, and assortment of dishes.

In the first two days, the patient's food requirements are negligible because of distressing precordial pains, general debility, and nausea. During this period it is preferable to restrict his diet to liquids given 6-8 times daily: one-fourth of a glassful of warm, weak tea with sugar, warm black currant juice, orange juice, sweetbrier water, sweet thin jellies. Unskimmed milk and grape juice are not recommended because they cause intestinal flatulence. Cold drinks are also forbidden as they are likely to cause pains in the heart region.

After the 3rd day of illness, in case of amelioration in the patient's condition, the diet for the first period of the disease is prescribed for a duration of 7-10 days.

Model Diet for the 1st Period of Disease

7-8 a.m. (on an empty stomach). Half-a-glassful of prune water. If the latter is unavailable, substitutes may be used, such as beetroot or carrot juice or sour milk.

8.30 a.m. Half-a-glassful of imitation coffee or tea with milk and 10 g of sugar, milk gruel, grated fresh apple with sugar (5 g).

11 a.m. Boiled chicken or cutlet, half-a-glassful of sweetbrier water.

2 p.m. Half-a-glassful of vegetable broth with egg flakes; boiled fish or quenelles; fruit jelly or half-a-glassful of kissel.

5 p.m. Apple purée or half-a-glassful of sour milk.

7 p.m. Mashed cottage cheese with sugar, half-a-glassful of sweetbrier water with 10 g of sugar.

9 p. m. Purée of 50 g of prunes.

The above diet contains: proteins, 40 g; fats, 25-35g; carbohydrates, 120-140 g. Caloric value: 800-1,000.

Model Diet for the 2nd Period of Disease

7-8 a.m. (on an empty stomach). Half-a-glassful of prune water.
8.30 a.m. Half-a-glassful of coffee or tea with milk, milk gruel, mashed cottage cheese or egg-white omelette.
11 a.m. Vegetable broth with egg flakes, rusks, boiled fish or chicken or steamed cutlet of 50 g of meat, apple or grape jelly.
5 p.m. Half-a-glassful of tea or juice or half-a-glassful of sour milk.
7 p.m. Boiled fish or chicken, carrot or beetroot purée or boiled cauliflower.
Before sleep. Purée of 100 g of prunes or half-a-glassful of sour milk.

The above diet contains: proteins, 50 g; fats, 30-40 g; carbohydrates, 180-200 g. Caloric value: 1,200-1,400.

With amelioration in the patient's condition a diet is prescribed for the third period of disease. Vegetables are served in purée form. The daily intake of common salt is 3-5 g, sugar 50 g, butter 30 g, white bread 150 g, liquid 800 ml (including liquid foods).

Model Diet for the 3rd Period of Disease

7-8 a.m. (on an empty stomach). Prune water or a glassful of sweetbrier water.
8.30 a.m. Buckwheat or any other ungrated gruel, lettuce, half-a-glassful of coffee or tea with 10 g of sugar.
11 a.m. Cottage cheese with one teaspoonful of sour cream, an apple or other fruit.
2 p.m. 200 g of borsch (beetroot and cabbage soup) or soup of grated vegetables, boiled chicken with loose rice or vegetable purée, half-a-glassful of fresh fruit compote.
5 p.m. Half-a-glassful of tea or sweetbrier water, cottage cheese with a teaspoonful of sour cream.
7 p.m. Boiled chopped meat with buckweat gruel, vegetable or fruit purée, half-a-glassful of tea.
Before sleep. 100 g of steeped prunes and a glassful of sour milk.

The above diet contains: protein, 7 g; fat, 50-60 g; carbohydrates, 200-220 g. Caloric value: 1,600-1,800.

From the sixth week a diet is prescribed for the fourth period of disease.

Model Diet for the 4th Period of Disease

7-8 a.m. (on an empty stomach). Prune water or a glassful of sweetbrier water.
8.30 a.m. Urgrated gruel, vegetable salad or omelette of 1-2 egg-whites.

11 a.m. 100 g of cottage cheese, 100 g of fruit, half-a-glassful of sweetbrier water.
2 p.m. 200 g of beetroot borsch or soup of ungrated vegetables, cutlets or a piece of meat with vegetable purée (roasted meat twice weekly), jelly, kissel, mousse, baked apple or compote.
5 p.m. Half-a-glassful of sweetbrier water, an apple.
7 p.m. Fish, chicken, or cottage cheese, carrot purée.
Before sleep. Steeped prunes or a glassful of sour milk.

The above diet contains: proteins, 90 g; fats, 50-70 g; carbohydrates, 250-300 g. Caloric value: 2,000-2,200.

This dietary pattern is usually followed during a month, gradually changing over to a diet recommended for coronary disease in the period without exacerbations.

If myocardial infarction runs a favourable course, strict confinement to bed for a period of 3-4 weeks is followed by measures to restore the patient's activity. From the 4th-5th week the patient may be cautiously turned to the right side and later is allowed to do it himself. From the 6th week he is permitted to sit up in bed. In the 7th-8th week the patient is allowed to rise from bed, and even walk a few steps about his ward. In the case of repeated infarctions the above-mentioned terms must be prolonged. The patient is discharged 8-10 weeks after the onset of the disease. He may be accommodated at a local cardiologic sanatorium in 4-5 months and return to work in 6-7 months.

ACUTE CIRCULATORY FAILURE

Normal circulation of the blood is ensured by a coordinated work of the heart and blood vessels. The amount of blood supplied into the aorta during a left ventricular systole depends on the quantity of blood entering the heart through the venous system. Acute circulatory failure is manifested by a steep fall in the arterial and venous pressure, a disturbed venous supply in the pulmonary circulation and a blood deficit in the peripheral vessels.

Acute circulatory failure is encountered with profuse haemorrhage, both external and internal, as well as with intoxications and infectious diseases. In such cases, the vascular tone is diminished, the venous bed is dilated, with a resultant steep decline in venous pressure, which is responsible for inadequate blood supply to the heart and, consequently, for a blood deficit in the arterial system. Acute circulatory failure also follows a large loss of fluid (in diarrhoea, vomiting).

In circulatory insufficiency, the circulating blood is reduced in volume, with a drop in the arterial and venous pressure. This is followed by a reflex contraction of the smaller arteries and veins owing to which the arterial pressure returns to normal. This ensures the blood supply to the vitally important organs: the heart, brain, kidneys, etc.

Treatment. A patient in the state of a shock or collapse is put into bed, his head being rested in a position lower than his feet to improve the blood supply of the brain. Hot-water bottles are applied to his feet. For an intense pain an injection of 1 ml of a 1 per cent morphine solution is helpful. In addition, the patient is given 100 g of alcohol and later is recommended to drink plenty of liquid. The principal task in the treatment of shock and collapse is to increase the amount of fluid in the body and raise the tone of the vascular system. For this purpose, the patient is given transfusions of blood or its substitutes in doses of 500-800 ml, injections of isotonic physiologic solution in a dose of up to 1 litre, and rectal drip of a 5 per cent glucose solution or physiologic solution. These measures are especially helpful in the case of a large loss of fluid due to vomiting or diarrhoea. The intravascular pressure is elevated by means of vasoconstrictors: a 0.1 per cent noradrenaline solution in a dose of 1 ml administered subcutaneously, a 5 per cent ephedrine solution in a dose of 1 ml, as well as camphor, sympathol, corasole, and a 10 per cent caffeine solution introduced in a dose of 1-2 ml subcutaneously, and a 1 per cent mesaton solution (formula No. 35*a*). Injections must be performed every 2-3 hours until an amelioration in the patient's condition, i.e., termination of the state of collapse or shock. Sometimes a subcutaneous injection of a 0.1 per cent strychnine solution in a dose of 2 ml is very efficacious.

CHRONIC CIRCULATORY FAILURE

With respect to the character of its course chronic circulatory failure is subdivided into three types: left ventricular failure, right ventricular failure, and combined cardiac failure involving both sides of the heart.

Insufficiency is manifested by symptoms of congestion in the pulmonary circulation in left ventricular failure, and in the veins of the general circulation in right ventricular failure. In combined left and right ventricular failure, congestive symptoms are noted both in the pulmonary and general circulation. The principal sign of cardiac insufficiency is inadequate arterial blood supply of individual organs, which leads to oxygen deficiency. Circulatory insufficiency is accompanied by a train of symptoms. Among them is deceleration of the blood flow, an elevation of the venous pressure, a disturbance in urinary secretion, etc.

The patient complains of breathlessness, palpitation, pains in the heart region, and a feeling of heaviness in the epigastrium. The causes of circulatory failure are defects in the heart valves and a lesion of the myocardium. These factors, however, are only some of those making up the mechanism of circulatory failure. A role of tremendous importance is played by neurogenous factors.

Left ventricular failure. This condition is observed in coronary sclerosis, atherosclerotic cardiosclerosis, hypertensive vascular disease, aortic valve insufficiency, syphilitic mesaortitis with stenosis of the orifice of the coronary vessels, infarction of the left cardiac ventricle, mitral valve insufficiency, and also in symptomatic hypertension.

The patient complains of shortness of breath during physical strain in the initial stages of the disease and later of permanent dyspnoea, and paroxysms of suffocation (cardiac asthma) occurring mostly at night. The above-mentioned symptoms are caused by inadequate blood supply of the brain, heart, kidneys, and other organs as a result of pulmonary congestion and excess blood in the pulmonary veins. Haemoptysis is a frequent occurrence. In the case of isolated left ventricular insufficiency there is no congestion in the liver and veins of the general circulation. The patient assumes an uneasy forced posture. When the left heart is only hypertrophied and there is no dilatation of its cavity, the apex beat may be intensified or elevating, while the heart has an aortic configuration. Later, when hypertrophy is joined by dilatation and the left venous orifice becomes distended, a relative mitral valve insufficiency ensues with a systolic murmur at the apex. Later, stasis develops in the pulmonary circulation with severe tachycardia, and the formerly clear accentuation of the second sound over the aorta becomes diminished. The inadequate muscular force of the left ventricle causes a reduction in the systolic pressure while the diastolic pressure remains unchanged. Impairment of the arterial blood supply of the brain produces convulsions, dizziness, loss of consciousness and a disturbance in the respiratory rhythm.

In attacks of cardiac asthma, a 1 ml dose of a 1 per cent morphine (pantopon, etc.) solution, preferably with 0.5 ml of a 0.1 per cent atropine solution, is administered in order to reduce the excitability of the respiratory and vasomotor centres, and in this way to dilate the bronchioles and reduce the tone of the pulmonary vessels. An attack is relieved within a matter of 5-10 minutes. For marked symptoms of circulatory failure (small pulse, low arterial pressure, etc.) morphine is injected simultaneously with camphor (cordiamine, cordiazol), as well as with 0.5 ml of strophanthin in 20 ml of a 40 per cent glucose solution administered intravenously. It is also recommended to administer 2-3 drops of a 1 per cent nitroglycerin solution dilating the coronary vessels, as well as euphyllin solution with 10 ml of a 20 per cent glucose solution, which dilates the brain and heart vessels. With considerable congestion in the pulmonary circulation blood-letting in quantities of 300-400 ml is undertaken. A similar effect is produced by the application of tourniquets to the upper and lower extremities for an artificial induction of a venous stasis in the periphery. Rubber tourniquets are applied to the base of the extremities at a medium pressure so as to prevent venous drainage but retain the inflow of arterial blood. This ensures a sharp reduction in the flow of venous blood to the heart and makes the patient's condition easier. The tourniquet has to be removed in 15-30 minutes.

Right ventricular failure occurs in such diseases where the right ventricle has to overcome increased resistance in order to pump blood into the pulmonary circulation. An isolated right ventricular failure results from destruction of the capillary network of the lungs in emphysema, pulmonary fibrosis, kyphoscoliosis, etc. Right ventricular failure may also be secondary to left ventricular failure with congestive symptoms in the pulmonary circulation and consequent difficulties in the performance of the right ventricle.

Right ventricular failure causes stasis in the vessels of the general circulation, hypertrophy and dilatation of the right ventricle, distension of the right fibrous ring and relative insufficiency of the tricuspid valve. The veins on the neck are swollen and throbbing. In the veins of the general circulation the blood flow is decelerated and venous pressure increased. An elevated pressure in the right ventricle causes blood congestion in the liver. The hepatic veins are short, devoid of valves, and therefore are very rapidly overfilled with blood. The enlarged congested liver is tender on palpation because of an acute distention of its capsule. There are sharp pains, vomiting and dyspeptic symptoms due to congestion of venous blood in the organs of the gastrointestinal tract. In tricuspid valve insufficiency there is systolic pulsation of the liver and veins on the neck. The liver becomes indurated and less sensitive on palpation (nutmeg liver); this is followed by ascitis.

Stasis extends to all organs connected with the portal system. There is oliguria and retention of common salt in tissues. Peripheral oedema appears, at first on the feet and shins, and later becomes generalized; the subcutaneous cellular tissue shows an accumulation of fluid (anasarca). Severe cyanosis is noted on external examination.

At the same time, there is frequent hypertension caused by stimulation of centres in the medulla oblongata by congested venous blood as well as by a circulatory disorder in the kidneys resulting in the secretion of vasoconstrictive substances. The very same congestive symptoms are often responsible for abnormalities in the central nervous system, such as delirium, psychosis, etc.

The heart sounds are dull. There is tachycardia and embryocardia, and a systolic murmur is heard over the xiphoid process of the sternum. When left ventricular insufficiency is joined by failure of the right ventricle, stasis in the pulmonary circulation comes to an end. The accentuation of the second sound over the pulmonary artery is diminished as the result of a lessened load on the pulmonary circulation and venous congestion in the veins and organs of the general circulation.

Combined cardiac failure. In a diffuse affection of the myocardium (myocarditis, cardiosclerosis, myocardial dystrophy, etc.), as well as when left ventricular failure is joined by failure of the right ventricle, signs of combined cardiac failure make their appearance. Congestive symptoms are present both in the pulmonary circulation and in the veins and organs of the general circulation. In combined cardiac failure, the signs of right ventricular insufficiency are especially manifest since the right ventricle pumps an insufficient quantity of blood into the pulmonary circulation, which makes the performance of the left ventricle much easier.

In the final stages of combined cardiac failure, fibrous tissue forms in the congested organs (brown induration in the lungs, sclerosis in the heart, nutmeg liver, etc.).

SYPHILIS OF THE AORTA (AORTITIS SYPHILITICA)

Syphilitic aortitis is one of the most frequent manifestations of visceral syphilis.

In syphilitic aortitis, a specific (gummatous) inflammatory process involves all layers of the aorta. The syphilitic lesion is predominantly localized in the initial (ascending) part of the aorta; therefore, the aortic valves and the ostia of the coronary vessels lying immediately under them are consecutively involved in the disease process. Simultaneously with destruction of aortic tissue and valves there is proliferation of coarse-fibre connective tissue in the damaged parts.

This results in the aorta losing its elasticityand distending, with deformation of the aortic valves, constriction of the ostia of the coronary vessels, which interferes with the blood circulation in the myocardium.

Far-advanced cases of syphilitic aortitis are characterized by anginal attacks occurring in combination with symptoms of aortic valve insufficiency: hypertrophy of the left ventricle, diastolic murmur over the aorta, a quick high pulse and paroxysms of cardiac asthma. The aorta is moderately dilated. In less advanced cases, the clinical features are less distinct and diagnosis of syphilitic aortitis is suggested by attacks of angina pectoris and aortalgia, i.e., prolonged pain in the region of the manubrium sterni, arising independently of physical exertion and failing to respond to nitroglycerin. The pains are concomitant with an insignificant dilatation of the aorta, a coarse systolic murmur and an accentuation of the second sound over the aorta. Such patients often exhibit signs of syphilitic lesions in other organs: the central nervous system, liver, and bones. In the majority of cases the Wassermann test is positive. Very important for the diagnosis of syphilitic aortitis is history taking, which helps reveal symptoms of syphilis sustained in the past: the presence of painless ulcers on the genitalia, periodically recurrent rashes, pains in the bones at night, spontaneous abortions, stillbirths, etc.

Aortic aneurysm (aneurysma aortae). Aortic aneurysm is secondary to syphilitic aortitis. As pointed out above, in the latter disease the aorta loses its elasticity and dilates under pressure from the blood.

In time, this dilatation grows in size. Rather than involve the entire aorta, it is usually found in parts where the damage is worst.

The clinical picture of the disease invariably includes the above-described symptoms of syphilitic aortitis, the only difference being a marked distention of the aorta. The distention is readily determinable on percussion; an intense pulsation of the aorta is felt on palpation in the jugular fossa. The x-rays reveal a sharp expansion of the aortic shadow. The distention is sometimes so large that the aneurysmatic sac is seen to bulge onto the anterior thoracic wall and, in inspection of the patient, is visible as a pulsating tumour. In other cases, the dilated aorta may compress the adjoining structures: the oesophagus, bronchus, recurrent nerve, brachial plexus, etc. This produces corresponding disturbances: a disorder in deglutition, retraction of the lung, a hoarse voice, neuralgic pains in the shoulder girdle. A very characteristic symptom of aortic aneurysm is discrepancy in the pulse on the right and the left hand.

Like in syphilitic aortitis, in aortic aneurysm the Wassermann test may be positive, which is a diagnostic sign.

The treatment of syphilis of the aorta involves considerable difficulties. A correctly instituted therapy, however, may ameliorate the course of the disease, ease the patient's condition and prolong his life. As regards drug therapy, it is recommended to take preparations of iodine and mercury as well as bioquinol, and penicillin preparations. Of the symptomatic remedies nitroglycerin is used for anginal attacks and strophanthin for attacks of cardiac asthma.

Over the past few years, surgery (excision) of the aortic aneurysm has been rapidly making progress.

The prognosis of a syphilitic affection of the aorta is always grave. Death follows the onset of symptoms of aggravating cardiac, less frequently coronary, failure. An aneurysm may cause death as the result of its rupture and a lethal haemorrhage.

Prophylaxis of syphilitic aortitis consists in a careful treatment of the primary forms of syphilis.

DISEASES DUE TO DISTURBANCES IN THE NERVOUS REGULATION OF VESSELS *

Vascular neurosis is the collective term denoting a large group of diseases originating from a disturbance in the nervous regulation of the vascular tone. Impaired innervation of vessels most frequently results from a disturbance in the normal interrelations between stimulation and inhibition processes in the central nervous system. Disturbances in the function of the central nervous system and, consequently, in the innervation of vessels may be due to a variety of causes: mental shock, unfavourable living conditions, certain endocrine diseases, various intoxications (uraemia), etc.

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* Hypertensive vascular disease belongs to the same group. In view of the fact, however, that later it produces organic changes, this disease is discussed in a separate section.

The cardinal clinical manifestation of vascular neurosis is an abnormally intense reaction of vessels to various stimuli. This may be attended with spasm or dilatation of vessels in a particular region.

A typical example of vascular neurosis is the so-called *Quincke's angioneurotic oedema*, a disease characterized by a sudden appearance of a localized, painless oedema of the extremities or of the face. It is treated by intravenous infusions of calcium chloride and x-ray irradiation of the posterior roots of the spinal cord. Another example of vascular neurosis is *Raynaud's disease*. It occurs as a spasm of the smaller arteries in the extremities (hands and feet). The spasm is attended with pain, marked pallidness and coldness of the fingers and toes. The disease runs a paroxysmal course and sometimes ends in gangrene of the fingers and toes.

Another disease in the group of angioneuroses is *migraine*. It is characterized by paroxysms of intense headache in some part of the head (frontal, occipital, temporal) and is accompanied by nausea, vomiting, salivation, and pallor. The simplest and highly efficacious remedy against migraine is the application of frequently changed hot compresses to the head. Aminopyrine and caffeine are also helpful. In certain cases, a good effect is produced by amyl nitrite and nitroglycerin.

Diseases of the Kidneys and the Urinary Passages

AN OUTLINE OF ANATOMY AND PHYSIOLOGY

The kidneys belong among paired organs. They are bean-shaped, each weighing about 150 g. The kidneys are extraperitoneal organs and lie along both sides of the vertebral column on a level with the 11th thoracic and 3rd lumbar vertebrae. Each kidney is enclosed in a dense readily removable capsule and is kept in place by retroperitoneal adipose tissue, the parietal peritoneum, and intraperitoneal pressure, but mainly by the short and strong vessels through which it communicates with the abdominal aorta and the inferior vena cava. The right kidney is situated somewhat lower than the left; during inhalation, both kidneys are displaced 4-5 cm downwards.

The kidney consists of two layers: a cortical (outer) and a medullary (inner) substance. The cortical substance, in which the glomeruli and tubules are lodged, is wedged into the medullary substance consisting of pyramids (Fig. 45).

In the cortical substance the renal artery divides into branches which form an extensive capillary network grouped in tufts known as Malpighian bodies, or glomeruli. Every glomerulus is invested by a capsule consisting of two membranes with a fissure between them. This capsule was for the first time described by M. Shumlyansky and 50 years later by Bowman.

Extending from the glomeruli are the proximal convoluted tubules which form the loop of Henle consisting of a descending and an ascending limb and ending in the distal convoluted tubule which terminates in the collecting tubule. The latter is continuous with the ureter, whose expanded inlet is known as the pelvis holding about 10 ml of urine. From the ureters the urine passes into the urinary bladder, whose holding capacity is 300 to 500 ml. The renal tissue contains about 2 million glomeruli which together with their investing capsules are equivalent to 50 sq m in area. The glomeruli have the function of filtration and formation of early tubular fluid (preliminary urine) (Fig. 46).

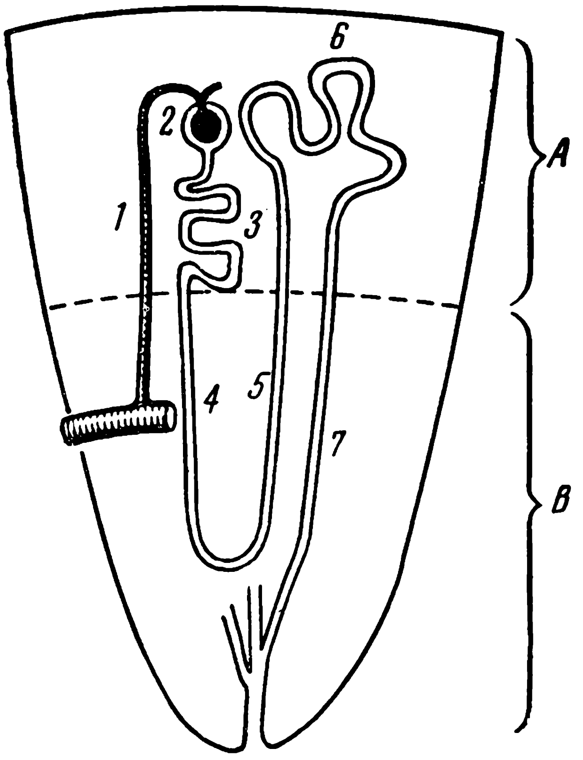

Fig. 45. Structure of the kidneys
A—cortical substance; *1*—interlobular artery; *2*—glomerulus; *3*—proximal convoluted tubule; *4*—descending limb of Henle's loop; *5*—ascending limb of Henle's loop; *6*—distal convoluted tubule; *7*—collecting tubule; *B*—medullary substance

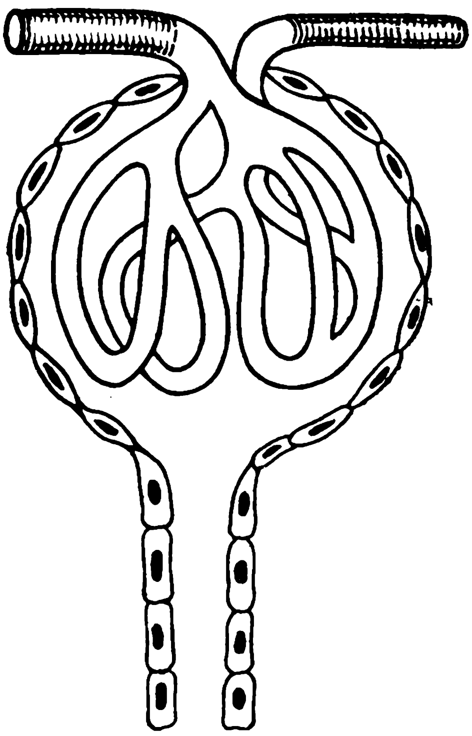

Fig. 46. Structure of glomeruli

The kidneys perform very important functions in the vital activity of the body. They eliminate the end products of protein metabolism, nitrogenous wastes and foreign matter, and regulate the arterial pressure and the acid-alkaline balance. The kidneys are the site of a very vigorous circulation of the blood: 700 ml of blood passes through them in one minute while the entire mass of circulating blood flows through these organs every 5-10 minutes. Such a vigorous circulation of the blood in the kidneys contributes to a rapid cleansing of the body from the end products of metabolism: urea, uric acid, creatinine, creatine, indican, organic and inorganic salts, foreign dyes and medicinal substances.

According to modern views, water and all solutes of the urine are filtered by the glomeruli; these solutes, however, are contained in the preliminary urine in the same concentration as in the blood. Later, the tubules absorb a considerable part of water (98 per cent of the total), sugar, the major part of common salt, and part of the urea, while creatinine and sulphates are not taken up at all. As a result of reabsorption the final urine is produced from the preliminary urine in the tubules. Owing to the contraction of the renal pelves and peristalsis of the ureters the urine is vigorously pushed forward and enters the urinary bladder which is periodically emptied 4-5 times daily in response to neuroreflex stimuli. Both the capillary network of the kidneys and the epithelial cells of the tubules are provided with a large number of nerve endings.

SYMPTOMS OF DISEASES OF THE KIDNEYS AND THE URINARY PASSAGES

The patient's complaints may be general and local in character. The latter are those of dull or acute pains in the small of the back in the presence of inflammatory symptoms in the kidneys, the pelves, and the perirenal cellular tissue. The complaints of a general character include debility, malaise, vomiting, diarrhoea, headaches, haemorrhages, and loss of vision caused by autointoxication with nitrogenous wastes. A patient with acute or chronic nephritis has a characteristic appearance: his face is pale and puffy, particularly in the presence of an extensive oedema.

Oedema. Oedema is one of the cardinal symptoms of acute and chronic nephritis and nephrosis. The causes of oedema are as follows: (a) retention of fluid and common salt in the tissue consequent upon a disturbance of filtration in the glomeruli and an intensified reabsorption of water and common salt in the tubules; (b) an increased capillary permeability due to an inflammatory process in the capillary walls; (c) a reduction in the protein content of the blood (hypoproteinaemia); (d) elevation of the intracapillary pressure causing an intensified filtration into the tissues.

All of the above-mentioned factors are acting asynchronously. In acute nephritis, the principal cause of oedema is a disturbance in the water-secreting function of the kidneys, which leads to retention of water and common salt in tissues. All other factors are of secondary importance. In amyloid-lipoid nephrosis and nephrotic forms of chronic nephritis the leading role in the formation of oedema is played by a derangement of water-salt metabolism and changes in the tubular system of the kidneys, which result in reabsorption through the tubules of a considerable part of common salt and water in the presence of normal glomerular filtration.

In order to determine whether oedema has increased or reduced one has to measure the daily fluid intake and urine discharge. This method, however, is quite unreliable; more accurate findings can be obtained by recording the patient's weight in the morning on an empty stomach. After the disappearance of visible oedema many persons with kidney diseases are sometimes found to have concealed oedema, and after abatement of the latter they for a long time have a marked predisposition to oedema, so the latter recurs in case of an excess intake of common salt and fluids.

Predisposition to oedema is determined by means of the so-called blister test; 0.2 ml of physiologic salt solution injected intracutaneously is fully absorbed in healthy persons within 45 minutes and in persons with predisposition to oedema within 15 to 20 minutes.

Renal oedema develops in characteristic places: in the early stage it appears in the loose cellular tissue of the eyelids, genitalia, and the scrotum, and later, by the force of gravity, in the lower parts, i.e., in the feet when the patient is standing and in the lumbar region when he is lying on his back. In massive oedema, which is the most frequent in nephrosis and nephrotic forms of nephritis, the fluid accumulates also in the serous cavities: the pleural, the abdominal and the pericardial.

Hypertension. Acute and chronic nephritis is attended with hypertension. Prolonged renal hypertension produces hypertrophy of the left cardiac ventricle and vascular sclerosis. With a normal renal blood flow, nephroses are not accompanied by hypertension. The latter appears only during shrinkage of the glomeruli due to amyloidosis.

Changes in the organ of vision. Prolonged hypertension in patients with chronic nephritis at first produces spasm and later sclerotic changes in the retinal vessels, which interferes with the normal nutrition of the retina.

Examination of the ocular fundus in such patients reveals hyperaemia and oedema of the optic papilla, constriction of the arteries of the ocular fundus and dilatation of its veins. In the final stages, the optic papilla and the macula lutea (yellow spot) are surrounded by lipoid infiltrates appearing as white foci.

Haemorrhages are fairly frequent. All these symptoms are usually grouped under the term *angiospastic neuroretinitis*.

The patient at first complains of a number of abnormal visual sensations: flying specks before the eyes, dimmed images of objects, the appearance of brilliant starlets in the field of vision. In the final stages, the loss of vision is often irreversible.

Proteinuria. Normally, the protein content of the urine is so insignificant as to be undetectable by conventional laboratory investigation. Sometimes, during sports competitions requiring considerable physical strain, in a circulatory disturbance of a variable degree, in case of symptoms of congestion in the kidneys, as well as in a febrile condition the urine may show the presence of protein (proteinuria). Prolonged proteinuria in the presence of other symptoms always indicates a kidney disease. Protein finds its way into the urine from the blood serum through the glomerular capsules, its filtration becoming possible only in case of a functional (venous stasis) or organic (nephritis and nephrosis) disorders in the kidneys.

Haematuria. Haematuria may be macroscopic or microscopic. The erythrocytes may be fresh and leached; the latter's presence is a sign of a renal origin of haematuria, whereas fresh erythrocytes may appear in the urine in case of affection of the urinary passages: the ureters, urinary bladder, and urethra. The urine voided may show the presence of blood in the beginning, at the end or during the entire act of urination. The presence of blood at the beginning of urination points to a lesion of the urethra; at the end, a lesion of the urinary bladder, and during the entire act of urination, to a renal origin of the disease. Renal haematuria may be bilateral or unilateral.

Pyuria. The presence of pus in the urine is a highly important diagnostic sign. From women urine should preferably be collected with a catheter to guard against the entry of leucocytes from the inflamed organs into the urine. Using a cystoscope and a catheter it is possible to ascertain whether the leucocytes have arrived from the urinary bladder or are due to a febrile condition.

The specific gravity of urine. Normally, the specific gravity of urine varies from 1.015 to 1.020. In chronic nephrosis, however, it may grow to 1.065, and in chronic nephritis, drop to 1.008-1.009 and remain at a constant level, a condition known as hyposthenuria (low specific gravity) and hypoisosthenuria (low specific gravity identical with that of blood serum).

The quantity of urine. Normally, the amount of urine discharged in 24 hours ranges from 1,000 to 1,500 ml, but in kidney disease variations may occur both toward an increase (polyuria) and a decrease (oliguria) in the amount of urine passed. Polyuria and oliguria are also encountered in absolutely healthy persons, and are connected with the season of the year and the dietary patterns. These abnormalities point to a kidney disease only when they are joined by other symptoms, such as hypoisosthenuria in polyuria, hypertension, hypertrophy of the left ventricle, the presence of blood and protein in the urine, changes in the ocular fundus, etc.

Complete suppression of urinary secretion in severe affections of the kidneys is known as anuria; retention of urine caused by a mechanical compression of the ureters or the urinary bladder is called ischuria; difficult and painful urination is termed dysuria.

Abnormally frequent and painful micturition in small portions (pollakiuria) is evidence of inflammatory processes in the urinary bladder or of a nervous disorder.

More copious urination at night compared to that in the daytime—nycturia—is characteristic of chronic nephritis in the period of insufficiency of renal function.

PHYSICAL METHODS OF EXAMINING THE KIDNEYS AND THE URINARY PASSAGES

Percussion and Palpation of the Kidneys

Percussion is performed by striking the region of the kidneys with a fist over the examiner's hand as well as by striking the lumbar region with the edge of a palm. The appearance of pains during the latter procedure will point to a positive Pasternatsky symptom. This symptom is encountered in nephrolithiasis, tuberculosis of the kidneys, inflammation of the perirenal cellular tissue and in pyelitis (inflammation of the kidney pelves).

The kidneys are accessible to palpation only when they are ptotic or enlarged. In lean persons, the right kidney which is situated lower than the left may in certain cases be palpable. Palpation of the kidneys is performed with the patient standing or lying down.

For palpation in a standing posture, the examiner takes his seat in front of the patient and places the flat of his left hand from behind on the region between the 11th thoracic and the 3rd lumbar vertebrae. The right hand is placed palm down at right on the anterolateral region of the abdomen, so that the finger tips are close to the rib margin. After this, the examiner attempts to bring his hands together, taking advantage of the patient's respiratory movements. At the maximum of deep inspiration the hands come into contact and seize the kidney. The latter's lower part is felt under the right hand as an elongated solid body. Palpation of the kidney is also effected by means of ballottement in which pushing strokes are made with the fingers of one hand in the direction of the other, making one feel as though these strokes are transmitted through the kidney. The strokes are made with the left hand from behind in the region of the last lower rib. An enlarged and considerably ptotic kidney is palpable by this method even when no connection exists with the respiratory movements. In such cases, the kidney is said to rebound.

For palpation of the kidneys in a recumbent posture (Fig. 47), the patient is enjoined to lie on his back. The examiner seated to the right of him places his hands in the same way as for palpation in a standing posture. The left hand is used to raise the small of the back toward the right hand, which is placed at the rib margin of the lateral part of the abdomen, and the kidney is palpated by applying finger pressure during inspiration. A considerably ptotic kidney may be palpated all over its area. It is easily determinable

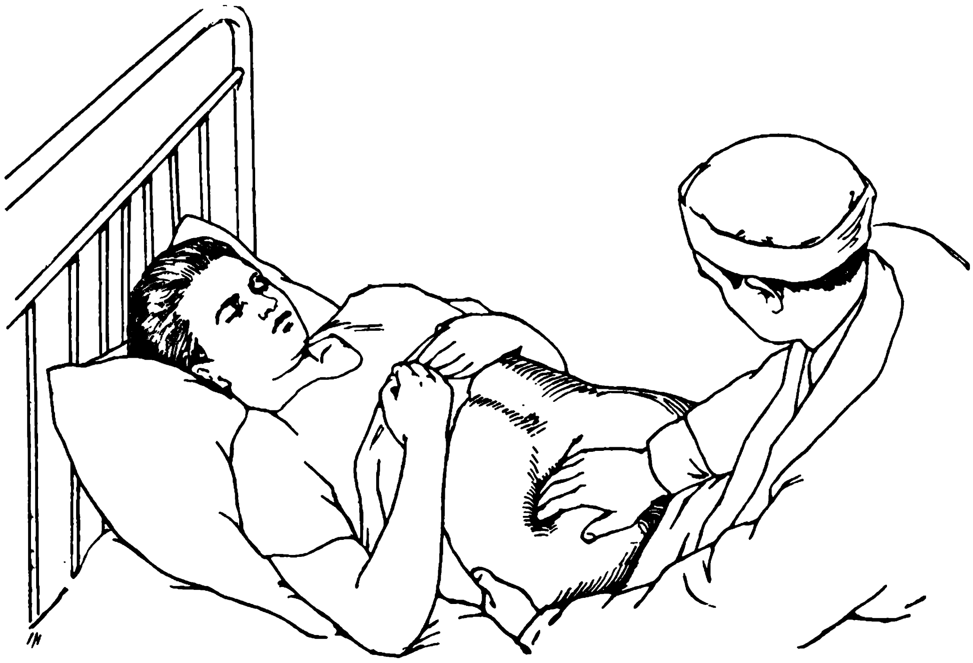

Fig. 47. Palpation of the kidneys

y its contours, since it is bean-shaped, has an elastic consistency nd a smooth surface. In case of insignificant ptosis, only the lower ole is palpable at maximum inspiration.

Considerable enlargement of a kidney occurs with tuberculous esions, newgrowths as well as with distention of the renal pelves vith retained urine or pus. The procedure for examination of the eft kidney is the same as that for the right.

Percussion of the Urinary Bladder

Dullness of the percussion sound over the pubis is noted in persons with an overfilled urinary bladder, which on palpation is felt as a round elastic body.

Functional Diagnosis of Kidney Diseases

The function of the kidneys is to eliminate water, solutes, nitrogenous wastes, common salt, and other substances from the body.

In clinical practice, the determination of the nitrogen-excreting function of the kidneys is of great practical importance since a reduction in the excretion of nitrogenous wastes points to a severe organic lesion. In the preliminary urine, the content of nitrogenous wastes is equal to that in the plasma, as a result of which its specific gravity is equal to that of the blood plasma also.

In a disturbance of the concentrating function of the kidneys compensation is attained in the early period by an elevated excretion of water, forced polyuria, which helps eliminate nitrogenous wastes from the body. The second stage of renal insufficiency is manifested by azotaemia due to a disturbance in renal filtration, when the quantity of urine progressively diminishes and azotaemic uraemia makes its appearance.

Azotaemia also develops in disorders of the renal circulation and diminution in the amount of urine, which take place in acute nephritis, cardiac diseases, etc. In such cases, azotaemia disappears after the blood flow in the kidneys has been restored to normal.

The specific gravity of urine is an index of the condition of renal function, because it mainly depends on the amount of water and urea and common salt solutes.

Water excretion. The water-excreting capacity of the kidneys is determined by means of the dilution test. The patient is given 1,500 ml of water of room temperature to drink within 30 minutes in the morning on an empty stomach after evacuation of the urinary bladder and measurement of body weight. Then the urine is collected once in every half-an-hour over a period of 4 hours, and the quantity and specific gravity of each portion are determined separately. Normally, the greater part of ingested liquid is excreted during the first two hours, and the remainder, at a later time. The specific gravity of urine is normally very low, ranging between 1.003 and 1.006. The water-excreting ability of the kidneys is considered inadequate in cases where the excretion of ingested liquid continues for hours, while the specific gravity of urine, which is low during the first two hours, fails to increase in the subsequent portions. The retention of ingested liquid is also determinable by measuring the body weight four hours after drinking the liquid.

Concentration test. The reabsorptive capacty of the renal tubular apparatus is estimated by keeping the patient on a dry diet. After a preliminary evacuation of the urinary bladder and measurement of body weight, the patient is forbidden to drink water for 24 hours and at the same time is given food rich in nitrogen (500 g of cottage cheese). The urine excreted is collected during 24 hours and the specific gravity of each portion is measured separately. Normally, a person kept on a dry diet excretes up to 700-800 ml of urine per 24 hours of a high specific gravity, reaching in certain portions 1.030 and more. In case of a disturbance in the concentrating capacity of the kidneys, the specific gravity falls short of 1.020 and in a high-degree insufficiency remains within the limits of 1.010-1.012.

The dilution test is contraindicated in disturbances of the renal blood circulation and in oedema, while the concentration test is harmful in azotaemia. The test, which can be undertaken regardless of the patient's condition, was suggested by the well-known Russian internist S. Zimnitsky. It is intended for a simultaneous determination both of the water-excreting and concentrating function of the kidneys. The Zimnitsky test is carried out as follows: in the morning, after evacuation of the urinary bladder, the patient is weighed and his urine is collected once in every 3 hours, from 6 a.m. to 6 a.m. of the next day (or from 8 a.m. to 8 a.m. of the next day). The first four portions collected from 6 a.m. to 6 p.m. constitute the diurnal urine excretion (DU); the remaining four portions collected from 6 p.m. to 6 a.m. of the next day, the nocturnal urine excretion (NU). The sum of diurnal and nocturnal secretions indicates the total urine secretion (TU) in 24 hours. When performing Zimnitsky's test, the patient is kept on his usual food and liquid regimen, note being taken of the amount of liquid intake. The quantity and specific gravity of urine are determined in each portion. The function of the kidneys is considered normal in all cases of substantial variations in the amount and specific gravity of urine in separate portions, depending on the quantity of liquid intake. A low and constant specific gravity of urine is a sign of a disturbed concentrating capacity of the kidneys. In a person with intact kidneys the specific gravity of urine in different portions may vary within a range of 1.010-1.025, the total amount of urine excreted must be equal to the amount of liquid intake, and the nocturnal urine excretion should not exceed one-third of the total.

Biochemical changes in the blood. Insufficiency of the concentrating function of the kidneys manifested by inadequate elimination from the body of the end products of metabolism, nitrogenous wastes in particular, gives rise to biochemical changes in the blood. In the process of enzymatic fission in the gastrointestinal tract the protein molecule breaks up into its principal components, amino acids, which undergo further dissimilation in the liver. This results in the formation of a number of nitrogenous wastes, such as urea, uric acid, creatine, creatinine, indican, etc., which are known by the collective name of nonprotein, or rest, nitrogen. Thus, nonprotein nitrogen is the entire blood nitrogen unconnected with protein. It is normally composed of urea, 20-40 mg/100 ml; uric acid, 2-4 mg/100 ml; creatinine, 1 mg/100 ml; indican, 0.05 mg/100 ml. Normally, the amount of nonprotein nitrogen varies within a range of 20-40 mg/100 ml, i. e., every litre of blood contains 0.2-0.4 g of nonprotein nitrogen. In persons with a deranged renal concentrating capacity the level of nonprotein nitrogen attains 100-200 mg/100 ml and climbs to higher figures very seldom. The level of nonprotein nitrogen is directly proportional to the degree of affection of the kidneys: the greater the damage to renal function, the higher the level of nonprotein nitrogen.

The amount of nonprotein nitrogen also increases during an intensified disintegration of protein substances (anaemia, suppurative processes in the lungs) as well as in cases of their impeded elimination as a result of a mechanical obstruction (compression of the ureters).

CLASSIFICATION OF KIDNEY DISEASES

Diseases of the kidneys have been known since ancient times, as is evident from literary sources. A classical description of renal disease, however, was for the first time given by the English physician Bright more than one hundred years ago. He also described the clinical symptoms of this disease: oedema, hypertrophy of the left ventricle, haematuria, proteinuria, headaches and pains in the lumbar region. This is why in many countries this malady is known as Bright's disease. Later,many authors described in detail various forms of kidney lesions and drew up their classification. For a long period of time, general recognition was given to the classification drawn up by the German authors Volhard and Fahr, who distinguished three principal groups of kidney diseases: (1) nephritis (inflammatory processes); (2) nephroses (degenerative processes); and (3) arteriolosclerosis of the kidneys. Later, various amendments and additions were proposed but until today there is no generally accepted classification of kidney diseases. In clinical practice, the classification presented below may be useful.

I. Nephritis (inflammatory processes in the glomeruli):

(1) Acute diffuse glomerulonephritis (acute nephritis);

(2) Chronic diffuse glomerulonephritis (chronic nephritis);
(3) Focal nephritis.

II. Nephroses (dystrophic lesions of the tubular apparatus of the kidneys):
(1) lipoid nephrosis;
(2) amyloid-lipoid nephrosis;
(3) necrotizing nephrosis.

III. Nephropathy of pregnancy.

IV. Nephroangiosclerosis.*

ACUTE DIFFUSE GLOMERULONEPHRITIS (GLOMERULONEPHRITIS DIFFUSA ACUTA)

Aetiology and pathogenesis. Acute diffuse glomerulonephritis, or acute nephritis, is in the majority of cases caused by streptococci, less frequently by pneumococci, which are harboured in the pharynx, in the upper air passages. Acute nephritis is secondary to tonsillitis, influenza, pneumonia, scarlet fever, endocarditis lenta, malaria. In rare cases, it may be due to other infections: diphtheria, tuberculosis, measles, infectious hepatitis (Botkin's disease), brucellosis, etc. Acute nephritis often develops after a common cold or a chilling of the body in persons working at a low or unstable temperature, which has prompted the conclusion that these factors are mainly responsible for the disease. In the majority of cases, however, chilling is only one of the factors contributing to the onset of nephritis, while the principal role is played by infection. Acute nephritis may also be caused by vaccination (serum nephritis) and certain medicinal substances (sulfa drugs, novarsenol, penicillin, etc.). In wartime the incidence of nephritis was fairly high; therefore, it was presumed that its precipitating causes are unhealthful dietary patterns, crowded surroundings, and infection. Soviet authors have established, however, that wartime (trench) nephritis, rather than being an independent disease, is of an ordinary infectious origin. Its development is especially favoured by chilling, bad weather in combination with mental and emotional strain.

Nephritis develops 10 to 20 days after infection. At this time, no pathogenic agents are found either in the urine or in the kidneys. On these grounds, nephritis has been regarded as an allergic reaction of the body to the entry of a foreign protein. The investigations carried out by V. Lindeman, N. Nefyodov and the Japanese scientist Masugi have proved the possibility of inducing experimental nephritis. Lindeman, for example, immunized guinea pigs with triturated rabbit kidney, after which the serum of these animals was inoculated to rabbits, and the latter died of uraemia in 3-5 days' time. Thus, it was proved that inoculation of renal tissue to guinea pigs is followed by the production of antibodies (immune bodies) which cause a disease similar to acute nephritis.

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* See Hypertensive Vascular Disease.

During the past few years, it has been possible to induce acute glomerulonephritis in rabbits by inoculation of streptococcic cultures. It was supposed that penetration of various disease-producing agents into the body, the kidneys in particular, causes a change in kidney proteins, as a result of which the latter become alien to the body, i.e., acquire the properties of autoantigens and provoke the formation of autoantibodies. The latter are responsible for the onset of diffuse glomerulonephritis. Consequently, while formerly the appearance of acute nephritis was attributed to the action of toxic substances produced by inoculation of a foreign protein, at present the development of acute nephritis is explained by an allergic reaction, which is caused by the action on the kidneys of the body's own proteins which have become alien (autoantigens) under the influence of bacterial flora and the formation of autoantibodies. Evidence in favour of the above-mentioned mechanism of origination of acute nephritis is a considerable increase in the titres of antistreptolysin and antihyaluronidase.

The vascular theory. Many authors (Volhard, P. Nikolayev, and others) assert that from the first day of its onset acute nephritis is accompanied by general angiospasm—spasm of the vessels of the skin, subcutaneous cellular tissue, and arterioles of the kidneys—only at a later time followed by inflammatory changes. Contemporary authors have established, however, that in acute nephritis the renal blood flow is undisturbed, and on the contrary, the glomeruli are hyperaemic, which is a sign of hyperergic inflammation.

Pathologic anatomy. Macroscopically, the kidneys are found to be of a normal size, colour and consistency, and their capsules peel off readily. Microscopy reveals symptoms of inflammation of the renal capillaries, enlargement of the glomeruli, distention and elongation of the capillaries, proliferation and desquamation of the epithelium. Haematogenous exudate with a large number of leucocytes accumulates in the capillaries. With respect to its morphological signs, acute nephritis is subdivided into the intra- and extra-capillary forms.

In the intracapillary form, the inflammatory changes are mainly confined to the glomerular capillaries; in the extracapillary form, to the lumen of the Shumlyansky-Bowman's capsule. This is the most severe form since it causes proliferation of the capsular cells with formation of crescents which compress the glomeruli. Inflammatory exudate in the capsular cavity may be serous, fibrinous and haemorrhagic in character.

Clinical picture. The common complaints are those of headaches, oedema, pain in the small of the back, scanty urination, breathlessness, debility and thirst. A highly important sign of acute nephritis is oedema. Oedematous swellings are localized on the face, the eyelids, less frequently they begin from the lower extremities. Sometimes no signs of oedema are seen on external examination because the fluid is accumulated in cavities. In certain cases, oedema grows so rapidly that within a short time the weight of the body increases by 10-15 kg and more. Sometimes, a massive oedema causes deformity of the torso and face. In bed-ridden patients, considerable oedema is noted in the small of the back and in the region of the genital organs. Oedema is very quickly followed by dyspnoea due to an elevation of the arterial pressure, cardiovascular insufficiency and general intoxication.

Hypertension is an inevitable and highly pathognomonic sign of acute nephritis. It may be brief in duration and unmarked, yet no acute diffuse nephritis is as a rule observed in the absence of hypertension. The arterial pressure is usually elevated: the systolic is within a range of 170-200 mm Hg; the diastolic, 100-120 mm Hg. In eclampsia the arterial pressure increase is larger. Arterial pressure returns to normal on the 10th-14th, less often on the 20th-25th day of the disease. Other symptoms are those of cardiac insufficiency: dilatation of the left cardiac ventricle, dyspnoea, palpitation, irregularity in heart beats, sometimes pains in the heart region, pains in the right hypochondrium, cough and blood-spitting. Later, the heart acquires an aortic configuration, with accentuation of the second sound over the aorta, a systolic murmur over the heart apex, dull cardiac sounds, and bradycardia. Right cardiac insufficiency is also observed sometimes.

The electrocardiogram shows left axis deviation (sinistrogram), changes of the *T* wave in all leads, diminished voltage, and sometimes extrasystolic arrhythmia. Acute nephritis is accompanied by haemodynamic changes: an elevated venous pressure, an increase in the volume of circulating blood to 8-9 litres (oedema of blood) and in the stroke volume of the heart, and an increased permeability of the capillaries which is attended with an intensified renal filtration in spite of the deceleration of the blood flow. The signs of cardiovascular insufficiency which appear in the early days of the disease aggravate during 2-3 weeks and gradually cease.

Bronchitis, bronchopneumonia, and pulmonary oedema are also noted. The gastrointestinal tract does not suffer substantial changes. Sometimes, there is nausea and vomiting of cerebral or renal origin, possibly occurring reflexly due to distention of the renal capsule. The liver is enlarged because of a diffuse oedema as well as in consequence of a disturbed blood flow.

There may be a change in the fundus oculi, though not at the onset of the disease, and symptoms of angiospastic retinitis make their appearance. The fundus is at first hyperaemic, the arteries and veins are dilated, which is followed by constriction of the retinal arteries, and at the site of intersection of the arterioles and venules the latter are found to be slightly stenosed (Gunn-Salus' sign).

The papilla is sometimes oedematous, its borders obliterated, with punctate haemorrhages in the retina.

Fever, leucocytosis, and an elevated ESR may be present only at the onset of the disease. Sometimes, acute nephritis gives rise to symptoms of haemorrhagic diathesis, nosebleed, Konchalovsky's tourniquet symptom, etc.

Changes in the blood and haemopoietic organs. In the majority of patients the blood is normal or hypochromic anaemia is present, which is possibly due to oedema of the blood. There is a likelihood of this anaemia resulting from a toxic affection of the bone marrow.

A characteristic sign of acute nephritis is eosinophilia, which may reach 15 per cent. There is a diminution in the total blood protein to 5-6 g/100 ml, a reduction in the level of albumins and an increase in α_2- and γ-globulins, as well as in fibrinogen. The diminution of the amount of albumins entails a reduction in the oncotic pressure, which is one of the causes of oedema. The sodium chloride content of the blood is increased to 600-700 mg/100 ml, the excretion of chlorides with urine is markedly reduced, but in the period of abatement of oedema is elevated, as a result of which their level in the blood is lowered.

Acute nephritis may be attended with mild azotaemia (500-80 mg/100 ml) which disappears early.

The pathology of urination is of decisive importance in identification of acute nephritis. The patient complains of pains in the lumbar region and dysuria. An important sign of acute nephritis is oliguria, which is sometimes preceded by anuria. In this condition, the urine is cloudy, reddish or dark-brown in colour, and sometimes even looks like meat slops. There is also macrohaematuria, and the specific gravity of urine is high. Proteinuria is one of the cardinal symptoms of acute nephritis, ranging within limits of 1 pro mille but also being likely to attain higher levels. It is especially pronounced in the early days of the disease, and its residual symptoms may linger for up to 6 months. The urine shows the prevalence of albumins, which constitute 60-90 per cent of protein, i.e., are close in composition to blood serum albumins.

Urinary sediment. The most important sign of acute nephritis is haematuria. It may terminate at an early time or last for months. Cylindruria is not inevitable. In many cases, only isolated hyaline or granular casts are found. During the past few years, the Kakovsky-Addis method has been used to determine the number of erythrocytes and leucocytes discharged with urine. Normally, the number of erythrocytes thus voided is about 130,000, and leucocytes, up to 650,000 in 24 hours. In acute nephritis, leucocytes vary in number from 15,000,000 to 1,340,000,000, and leucocytes, to 40,000,000 (Fig. 48).

The forms of acute nephritis. This disease is known to occur in a haemorrhagic or an oedematous-hypertensive form. The former

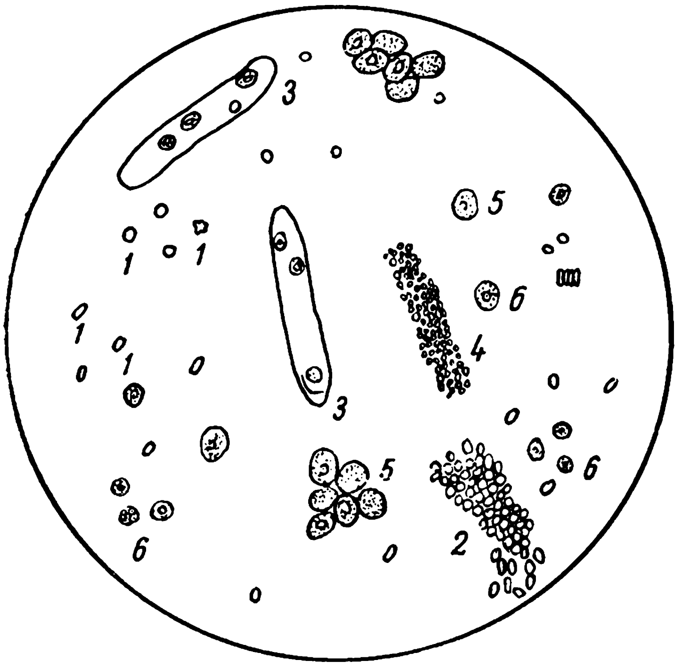

Fig. 48. Microscopy of the urine in acute nephritis

1—erythrocytes; *2*—accumulation of erythrocytes; *3*—hyaline casts; *4*—granular casts; *5*—renal epithelium; *6*—leucocytes

is characterized by pains in the small of the back, headaches and marked haematuria, proteinuria, and cylindruria. The latter form runs its course with headaches, oedema, hypertension, dyspnoea, proteinuria, cylindruria and mild haematuria.

The functional capacity of the kidneys. In acute nephritis glomerular filtration is impaired, reabsorption of the filtrate in the renal tubules is increased; this is followed by oliguria. The urine concentration is, as a rule, unchanged.

The condition of the central nervous system. In certain cases, drastic changes take place in the central nervous system, such as renal eclampsia or angiospastic encephalopathy. Renal eclampsia appears without precursory symptoms or after a preeclamptic period (intense headaches, elevated arterial and cerebrospinal pressure). Other symptoms are at first tonic and then clonic spasms of the smaller muscles, slanting of the eyeballs, biting of the tongue, foaming at the mouth, absence of reaction to light in the pupils, loss of speech, and motor unrest. There is confusion of consciousness and torpor. The convulsive state lasts five minutes to half-an-hour, and is followed by a brief period of relative calmness. During an attack, the pulse is slowed down, while the arterial and cerebrospinal pressure is increased to 350 mm of water. The tendon reflexes are increased, with a positive Babinski's sign. There is also a transient blindness with no change in the fundus oculi. Sometimes, termination of convulsions is followed by a state of excitement lasting for 24 hours and longer. Attacks may recur from two to five times daily. They gradually decrease in intensity and cease just as suddenly as they began.

Wartime nephritis. This disease runs a more severe course than common nephritis. Its cardinal symptoms are considerable oedema, collection of fluid in cavities, and marked disorders in the cardiovascular system. The urine shows massive proteinuria and macrohaematuria. More often, an elevation of body temperature is noted and the disease is sometimes complicated by renal eclampsia.

Outcomes of acute nephritis. During the past few years, fatal outcomes have been seen in not more than 0.3-0.4 per cent of cases.

and have been due to a heart failure, pneumonia and other infections, less frequently, to cerebral haemorrhage and azotaemic uraemia.

Differential diagnosis is not difficult when acute nephritis is running its typical course. Sometimes, great difficulties are likely to arise in differentiation between acute and chronic nephritis. In such cases the diagnosis is verified by history taking and examination of the cardiovascular system. Hypertrophy of the left ventricle is more frequently observed in exacerbation of chronic nephritis. In acute nephritis, anaemia develops in the period of oedema, and in chronic nephritis it progresses irrespective of oedema and polyuria. Acute diffuse nephritis must be differentiated from focal nephritis. The latter is characterized by the presence of the principal disease (subacute bacterial endocarditis, focal infection, cystitis, pyelitis, etc.), and by absent hypertension. Oedema and haematuria, even in the presence of hypertension, are not always sufficient evidence in favour of a diagnosis of acute nephritis, since oedema may be found with heart disease and haematuria, with a congested kidney, tuberculosis and tumour of the kidney, haemorrhagic diathesis, etc.

It should be borne in mind that proteinuria develops not only in acute nephritis but also in many pathologic states: in the premenstrual period, concussion of the brain, shock, postepileptic attacks, leucosis, etc. In certain cases, nephritis contributes to the clinical picture of periarteritis nodosa, subacute bacterial endocarditis, disseminated lupus erythematosus, and may camouflage the principal disease. Nephritis must be differentiated from urologic diseases. The latter are accompanied by pains in the small of the back, dysuric symptoms, etc.

Prognosis. Acute nephritis runs a severe course in elderly persons, men in particular. In hypercholesterolaemia, prognosis is worse because of a possible dystrophy of the tubular apparatus. Inadequate restoration of the kidney functions (filtration and absorption) during six weeks is a formidable sign. Recovery is facilitated by early hospital treatment. Confinement to bed is indispensable during 4-5 weeks, while hospital treatment should be continued for at least 2 months, temporary disability lasting from 2 to 3 months. Upon recovery persons working in damp surroundings and exposed to the risks of soaking and chilling should change their job.

All patients recovered from acute nephritis should be kept under medical observation at the local clinico-prophylactic centre (dispensary).

Prophylaxis necessitates the following measures: (1) prevention of infection (catarrh of the upper air passages, pneumonia, tonsillitis, etc.) and its early elimination; (2) general invigoration of the body by means of sports and physical culture; (3) treatment of carious teeth, treatment of chronic tonsillitis; (4) prevention of chilling of the body, and (5) desensitization of the body with dimedrol, calcium salts, etc.

Treatment consists in complete bed rest and uniform application of heat. For a large oedema a regimen of fasting and thirst is advisable after a preliminary intravenous infusion of 10 ml of a 25 per cent magnesium sulphate solution and administration of saline purgatives; diuretics (novurit, phonurit, etc.) may also be used. The arterial pressure is lowered with reserpine, while strophanthin, caffeine, and cordiamine are used for cardiovascular insufficiency, blood-letting, for cardiac asthma. To eliminate ischemia of the kidneys in the presence of marked oliguria, especially anuria, diathermy is applied to the kidney region (if there is no pronounced haematuria), as well as perirenal novocain block, decapsulation, and denervation of the kidneys.

Dietotherapy is an important remedy against acute nephritis. The diet must contain not more than 2-3 g of common salt. On definite days only sugar is given (200-300 g of sugar and 1.5 litres of water). This promotes diuresis and has a favourable effect on the function of the heart and kidneys. A diet rich in calcium salts contributes to induration of vessels, diminution of inflammatory exudation and an increase in blood coagulation. A diet of potatoes, apples and fruit is also prescribed. It contains 1,500 g of potatoes, 75 g of butter, and 500 g of sugar-sprinkled apples. This diet is very low in common salt (1 g) but is rich in potassium salts (20 g) and proteins (25-30 g), while apples also produce a diuretic effect. It is preferable to use milk and egg rather than meat protein, since the latter contains extractive substances irritating the kidneys. Upon amelioration of the patient's condition, the quantity of protein in his diet may be increased.

Renal eclampsia is relieved by blood-letting, lumbar puncture and introduction of chloral hydrate into the rectum. The intracranial pressure and cerebral oedema are reduced by intravenous drip of 100-150 ml of a 10 per cent magnesium sulphate solution. intravenous injection of 20-40 ml of a 40 per cent glucose solution, and perirenal novocain blocking.

For protracted anuria an artificial kidney is used. The remedies against chloropenia are intravenous infusion of 20 ml of a 10 per cent sodium chloride solution, salted food and profuse liquid intake, vitamins K and C, and rutin.

CHRONIC DIFFUSE GLOMERULONEPHRITIS (GLOMERULONEPHRITIS CHRONICA DIFFUSA)

Chronic nephritis, otherwise known as chronic diffuse glomerulonephritis, is an inflammatory infectious allergic disease.

Aetiology. In the majority of cases, chronic nephritis is secondary to inadequately treated acute nephritis, although the patient's history of past diseases often contains no evidence of acute nephritis, since the latter may have run such an imperceptible course as to have been completely forgotten by the patient. In some cases, a patient with chronic nephritis reports a history of focal nephritis. It is possible, however, that the latter had in fact been an obliterated form of acute diffuse nephritis which subsequently took a chronic course.

Chronic nephritis is due to the same aetiologic factors as those responsible for acute nephritis, i.e., bacterial infection (streptococci, etc.). The causes of transition of acute nephritis into a chronic form are variable. They are as follows: (a) belated and inadequate therapy; (b) the presence in the body of a chronic focus of infection (in the tonsils, carious teeth, accessory nasal sinuses, gallbladder, etc.); (c) unhealthful working and living conditions entailing frequent physical fatigue, chilling concurrent with infection, etc. Patients with a history of acute nephritis possibly retain autoantibodies in the blood, which sustain the inflammatory process in the kidneys and contribute to the transition of the acute disease into a chronic one.

In the majority of cases, chronic nephritis is due to a prolonged action of infectious foci present in the body, which cause sensitization, hyperergic inflammation in the glomeruli with an increase in their permeability and later also severe anatomophysiologic changes in the kidneys. In chronic nephritis the tubules also suffer changes more frequently than in acute nephritis.

Chronic nephritis is also encountered with systemic lupus erythematosus, subacute bacterial endocarditis and rheumatic fever; in these diseases, however, nephritis is only one of the accompanying manifestations of the principal disease—collagenosis.

Pathologic anatomy. Macroscopic and microscopic changes in the kidney depend on the duration and intensity of the disease process. The weight of the kidney is increased two-fold as compared to normal. The kidney is enlarged and white in colour. It is observed in chronic nephritis with a nephrotic component. Later, when the glomerular contraction processes join the clinical picture, the kidney diminishes in size, its surface becomes granular, and the capsule peels off with difficulty. This is the so-called secondary contraction of the kidney, i.e., its diminution following its enlargement. The microscopic changes correspond to the developmental phases of chronic nephritis. In the early stage of the disease in the intracapillary form one can see enlarged glomeruli with proliferation of the endothelium and degenerative changes in the tubules. The extracapillary form is characterized by marked proliferation in Shumlyansky-Bowman's capsule, the external membrane showing proliferation of the endothelial cells which in the form of crescent structures compress the glomeruli, thereby disturbing their function. A fatty or lipoid degeneration of the tubules is noted. In a kidney suffering secondary contraction the glomeruli and tubules are devoid of contents, the epithelium of Shumlyansky-Bowman's capsule becomes atrophied, the lumen of the smaller arterioles is narrowed or completely obstructed. In the nephrotic form, lipoids are deposited in the epithelium of the tubules, the latter become swollen and their walls undergo granular degeneration.

Clinical picture. In respect of its clinical course, chronic nephritis is known to occur in several forms: (a) latent; (b) oedematous-proteinuric; (c) hypertensive; (d) oedematous-hypertensive.

The clinical picture of chronic nephritis is subject to extensive variations depending on its forms.

In a latent form, nephritis progresses slowly, at first causing little discomfort to the sufferer and manifesting itself only by proteinuria and haematuria. Infrequently, the patient complains of debility and pains in the small of the back. This form of chronic nephritis runs a prolonged course and is, as a rule, benign in character, the patient retaining his capacity for work. Sometimes, this form may run its course with frequent exacerbations, in which case the latent form changes into an oedematous-proteinuric or hypertensive form.

The oedematous-proteinuric form of chronic nephritis gives rise to considerable oedema, including accumulation of fluid in the serous cavities, pronounced proteinuria, hypercholesterolaemia, moderate haematuria, and hypertension. The protein content of the blood is reduced to 5-6 g/100 ml. The urine shows the presence of lipoids and its total amount passed in 24 hours is diminished. The disease process is in the main intracapillary, though without any marked arteriolosclerosis. In the terminal stages oedema begins to subside and disappears completely. This, however, is followed by an arterial pressure elevation, and gradual progression of uraemia to a fatal outcome. This form of chronic nephritis is the most frequent one.

A patient with a hypertensive form complains of headaches, muscle spasms, impairment of vision, and an alimentary disorder. There is a considerable elevation of the arterial pressure to 200/100-220/120 mm Hg, whereas oedema is absent. The urine shows mild proteinuria and haematuria. Further progression of nephritis is accompanied by dyspnoea and tachycardia as a result of hypertrophy of the heart muscle due to a high elevation of the blood pressure, as well as degenerative changes in the myocardium. Later, the insufficiency of renal function grows worse, the blood shows an increase in the level of urea, indican, creatinine, and nonprotein nitrogen, the patient dying of azotaemic uraemia.

Extracapillary nephritis. Subacute, or extracapillary, nephritis deserves special description. This form runs a malignant course, the disease process tending to progress and cause severe lesions of the glomeruli and tubules with a disturbance in renal function. There s also isosthenuria and hyposthenuria, the blood showing an increase in urea, creatinine, indican and nonprotein nitrogen levels. The arterial pressure persists at a high grade and oedema fails to diminish.

Highly important signs of chronic nephritis are changes in the urine (proteinuria, haematuria), oedema, hypertension, cardiac failure, anaemia, abnormality in the fundus oculi, and azotaemia. Depending on the type of chronic nephritis, duration and intensity of the disease process, some of the above-mentioned symptoms may be absent or present in a mild or marked form.

Changes in the urine. Proteinuria is one of the principal symptoms of chronic nephritis. The protein content of the urine varies in the large: from tracer amounts to 10 pro mille and more. Massive proteinuria is observed in the oedematous-proteinuric form of the disease. In this condition, the urine, in addition to albumins, contains α_2- and β-globulins, and in the terminal phase, also γ-globulins. In the hypertensive form, the urine primarily contains albumins.

Haematuria is one of the cardinal symptoms of chronic nephritis. Considerable haematuria, just as a high-degree proteinuria, is not always an unfavourable prognostic sign. In the terminal phase, the amount of protein and blood corpuscles may be negligible due to the death of glomeruli, with an obviously inadequate function of the kidneys and azotaemia.

The urine may show the presence of many tube casts and leucocytes, particularly in the oedematous-proteinuric form. Up to 50,000,000 erythrocytes and 1,000,000 tube casts are excreted with the urine in 24 hours. In the oedematous-proteinuric form, there is a large number of epithelial cells which have undergone lipoid degeneration, as well as birefractive substances (lipoids).

Oedema is found mainly with the oedematous-proteinuric form, whereas in the latent and hypertensive forms it is present in exacerbations. In the terminal phase, oedema may be cardiac in origin.

Arterial hypertension is a permanent and frequent symptom of chronic hypertensive and oedematous-hypertensive nephritis. It tends to aggravate with the progression of the disease. Almost every type of chronic nephritis is accompanied by hypertension, which may show a reduction, although never reaching the normal level, as distinct from hypertensive vascular disease. Stable diastolic arterial pressure within the range of 120-130 mm Hg is an ominous prognostic sign.

The cardiovascular system is also affected in chronic nephritis, particularly when it is running a protracted course. The usual symptoms are hypertrophy of the left ventricle, muffled cardiac sounds, a systolic murmur often heard over the heart apex, accentuation of the second sound over the aorta, and a rare and tense pulse. Attacks of cardiac asthma, gallop rhythm and extrasystole may also be observed. After the onset of right ventricular insufficiency, the liver grows in size and is tender on palpation. The electrocardiogram shows a sinistrogram, sometimes a dextrogram, the *T* wave is flattened or biphasic in leads I and II, the *QRS* complex is expanded.

Anaemia is normochromic, normocytic, less frequently macrocytic and hypochromic, and is a permanent and characteristic symptom of great prognostic value. It is caused by dyspepsia and by the toxic influence of nitrogenous wastes on the bone marrow. The white blood does not suffer substantial changes. Only a slight neutrophilic leucocytosis is observed sometimes. Haemorrhages depend on a lesion of the vessel wall as well as thrombopenia or are associated with other factors of the blood coagulation system.

Angiospastic retinitis is mainly found in persons with chronic nephritis running its course with a high arterial pressure. The usual symptoms are constriction of the arteries of the fundus oculi, dilatation of the veins, Gunn-Salus' sign, the so-called copper or silver wire symptom, oedema of the retina or optic papilla, as well as haemorrhages of a variable degree. Sometimes, there is detachment of the retina with a large loss of vision, even to the degree of total blindness. The survival after the onset of angiospastic retinitis is from one to two years. Angiospastic retinitis is caused by stenosis of the arterioles of the fundus oculi and elevation of the intracapillary and intracranial pressure.

Chronic nephritis may be accompanied by various complications: sepsis, erysipelas, pneumonia, purulent infections, and cardiovascular insufficiency. The most formidable complication, however, is azotaemic uraemia resulting from a disturbance in renal function, above all the concentrating and later the excretory function.

The first sign of secondary contraction of the kidney is polyuria with a simultaneous abatement of oedema. The patient discharges 2 to 3 litres of urine of a low specific gravity daily, thereby increasing to a certain extent the excretion of nitrogenous wastes (compensatory polyuria).

AZOTAEMIC URAEMIA

Azotaemic uraemia is manifested by a functional disturbance in various organs, systems and the body as a whole. The patient complains of absent appetite and sometimes of aversion to food, especially protein food, a feeling of heaviness in the epigastrium, nausea and vomiting. The tongue is coated and dry, the breath smells of urine due to excretion through the gastric mucosa of urea which, under the action of bacteria of the oral cavity, is converted into ammonium carbonate. Another symptom is diarrhoea, which is sometimes bloody. The above-mentioned symptoms are caused by a compensatory excretion through the stomach and intestine of nitrogenous substances accumulated in the blood, which irritate the mucous membranes of the stomach and intestine and produce excretory gastritis and ulcerative-haemorrhagic inflammation of the intestine.

The respiratory organs show a proneness to the development of bronchitis. Respiration is heavy and loud, inspirations becoming deep and prolonged, and expirations, brief. This is caused by stimulation of the respiratory centre by acids accumulated in the body (acidosis). Sometimes, uraemia is attended with Cheyne-Stokes' respiration as a result of a sharp decrease in the excitability of the respiratory centre due to sclerotic changes in the brain vessels. In uraemia the vision markedly deteriorates, even to the degree of total blindness. At first the patient complains of haziness before the eyes. Later he becomes unable to recognize persons though his photoreception is retained. Finally he becomes totally blind in both eyes. Some persons have an impaired hearing and severe itching, which tends to grow in intensity parallel with aggravation of azotaemia. Shortly before death, nitrogenous wastes (urinary sweat or urhidrosis) are deposited on the facial skin, the wings of the nose and forehead.

Uraemia is accompanied by profuse haemorrhages, more frequently from the nose, and extravasations into the skin due to an injury to the walls of smaller vessels. The blood shows pronounced anaemia and leucocytosis, which tend to aggravate with the progression of uraemia.

In the terminal stages of the disease, azotaemic pericarditis develops, with a pericardial friction sound at the base of the heart. Since pericarditis appears shortly before death this friction sound is called a "funeral knell". Before death patients with azotaemic uraemia often develop azotaemic pneumonia, which is very difficult to identify because it runs its course at normal body temperature.

Azotaemic uraemia is accompanied by a severe functional disturbance in the central nervous system due to intoxication. The patient complains of persistent headaches, dizziness, growing debility, sleepiness, apathy and loss of working capacity. He is half-unconscious, drowsy in the daytime, and suffers from insomnia at night.

Uraemic coma is the final stage of autointoxication. In this condition, tonic spasms occur, mainly in the gastrocnemius muscles, and smaller clonic spasms and twitching of tendons in the muscles of the limbs and face are encountered. Severe paroxysms of convulsions are observed less often. In the second stage of renal insufficiency, there is a large loss of working capacity. A patient with severe azotaemic uraemia is forbidden to work, particularly in the presence of neuroretinitis.

Prophylaxis and treatment. Cases of radical recovery from chronic nephritis are rare; therefore, the principal goal is to prevent a rapid progression of the disease. For this purpose, elimination of infectious foci (tonsillitis, maxillary sinusitis, otitis, etc.) is undertaken. An important role is played by a therapeutic regulation of diet. In case of marked oedema, the intake of salt is restricted to 2-3 g per 24 hours, in severe cases, to not more than 1 g a day. A milk diet is recommended, which contains sufficient proteins, fats, carbohydrates, vitamins, and not more than 1-2 g of common salt per litre of milk.

A patient with an oedematous-proteinuric form of chronic nephritis is kept on a diet containing 2 g of rich proteins (meat, fish, cottage cheese, etc.) per kg of body weight.

In the presence of hypoproteinaemia, particularly that occurring in combination with anaemia, transfusions of whole blood, plasma and albumin substitutes are helpful.

In all cases of derangement of nitrogen excretion by the kidneys, the proteins in food must be strictly limited, or even completely excluded from the diet during uraemia. Carbohydrates and fats are adequately tolerated by patients, therefore, no restrictions on them are necessary. The diet in uraemia should be composed of fruit, compotes (stewed fruit) and kissel (thin jelly). Other remedies for uraemia are blood-letting in amounts of 400-500 ml (in the absence of marked anaemia), alkaline mineral water per os, enema of one teaspoonful of soda to a half-glassful of water, aspiration of gastric juice through a thin tube, lavage of the stomach through a thick tube, and administration of aminazine for vomiting.

For pronounced oedema diuretics are used, such as novurit, phonurit, as well as hypothiaside, especially in the presence of concomitant hypertension.

A patient with an oedematous-proteinuric form of the disease is given ACTH in doses of 40-60 units during a period of 10-15 days, and prednisolone in doses of 10-15 mg in 24 hours during 2-3 weeks. If abatement of oedema is accompanied by an elevation of the arterial pressure and an increase in haematuria, hormone therapy must be combined with the administration of hypotensive drugs: reserpine, dibazol, magnesium sulphate, etc.

A beneficial effect is produced also by the application of heat improving the blood flow in the kidneys (diathermy, UHF currents, "Sollux" light). In aggravation of haematuria these therapeutic measures are discontinued. For anaemic conditions, vitamin B_{12}, iron preparations and blood transfusions are prescribed.

Patients with an oedematous-proteinuric form of the disease as well as with moderate hypertension in the presence of the initial symptoms of renal insufficiency are advised to take a course of treatment at a climatic resort.

The prophylactic measures are the same as those used for acute nephritis. Chronic nephritis should be differentiated from acute nephritis, focal nephritis, hypertensive vascular disease and amyloid-lipoid nephrosis.

FOCAL NEPHRITIS (NEPHRITIS INSULARIS)

Focal nephritis is characterized by an affection of some of the renal glomeruli, and is not accompanied by symptoms characteristic of diffuse nephritis, i.e., hypertension, hypertrophy of the left cardiac ventricle and oedema. The only clinical manifestations of focal nephritis are mild changes in the urine.

Aetiology and pathogenesis. Focal nephritis is caused by microbial flora penetrating into the renal tissue with the blood stream. This is observed in exacerbations of a chronic focal infection harboured in the tonsils, teeth, accessory nasal sinuses, gallbladder, vermiform appendage, etc.

In septic processes, in subacute bacterial endocarditis in particular, tiny infected emboli are brought into the renal glomeruli with the blood stream and give rise to focal embolic nephritis.

The body gives an allergic reaction to the invasion of renal tissue by pathogenic microorganisms. At the same time, necrosis and thrombosis of the capillaries are noted. Degenerative changes occur in the tubules. Later, the glomeruli may become empty, undergo hyalinosis or be displaced by connective tissue, with destruction of a group of tubules.

Clinical picture. The patient, with rare exceptions, gives no complaints of a kidney affection, because symptoms of the principal disease come to the foreground. The cardinal symptoms of focal nephritis are a slight pain in the small of the back, microhaematuria, insignificant albuminuria and slight cylindruria. The urine sediment may also show the presence of epithelial cells. Hypertension, hypertrophy of the left cardiac ventricle, oedema, disturbance of renal function are usually absent in focal nephritis and may appear only when the latter has developed into diffuse nephritis.

The *diagnosis* of focal nephritis is, in the majority of cases, accurate, since it is based on a study of the causal relationship between a number of diseases and the concurrent focal nephritis. Difficulties are sometimes encountered in making a differential diagnosis between focal and diffuse nephritis, especially in case the latter disease runs its course without oedema and hypertension.

Prognosis in septic focal nephritis is ominous and depends on the principal disease, i.e., septis. In other cases, where the origination of focal nephritis is associated with chronic focal infections (tonsillitis, dental caries, inflammation of the paranasal sinuses, etc.), focal nephritis is not particularly detrimental to health and passes along with recovery from the principal disease.

Treatment. This consists in administrations of various antibiotics and elimination of all foci of infection in the body. Antiallergic

NEPHROSES

Nephroses, or kidney dystrophy, are due to a variety of causes, and are accompanied by degenerative changes in the tubules and severe metabolic disorders, i.e., in interchange of water and salts, proteins, cholesterol, etc.

Today nephroses are subdivided into the following groups: (1) lipoid nephrosis; (2) amyloid-lipoid nephrosis; (3) necrotizing nephrosis; (4) nephropathy of pregnancy, otherwise known as late toxaemia of pregnancy.

Lipoid Nephrosis (nephrosis lipoidea)

Aetiology. Lipoid nephrosis is a rare disease. In the majority of cases it is due to chronic infections: tuberculosis, syphilis, chronic purulent diseases of the lungs, osteomyelitis, etc. A number of authors trace the origin of lipoid nephrosis to dysentery, chronic enterocolitis, diphtheria, polyarthritis, chronic intoxication with heavy metals and neosalvarsan, prolonged administration of mercurial diuretics, and lymphogranulomatosis. In many cases, however, the aetiology of lipoid nephrosis remains obscure.

Pathogenesis. Until recently, degenerative changes in the renal tubules were believed to be a secondary affection caused by a functional disturbance in the thyroid gland and the adrenals, with ensuing passage of proteins through the kidneys into the urine. An analogy was drawn between this disease and diabetes mellitus, therefore lipoid nephrosis was otherwise known as diabetes albuminicus. Today, however, lipoid nephrosis is considered a primary disease. Nevertheless, some investigators assert that nephrosis, rather than being an independent disease, is a variety of nephritis.

Pathologic anatomy. The kidneys are enlarged, the capsule peels off easily, and the surface is smooth and pale-grey in colour (so-called large white kidney). In microscopy, some of the tubules are atrophied while others are swollen, with signs of granular, hyaline, and vacuolate dystrophy. The tubules harbour hyaline, granular and waxy casts in their lumina, and the epithelium of the tubules contains birefractive substances of lipoid origin (cholesterol esters). Analogous changes may be found in the glomeruli and the epithelium of Shumlyansky-Bowman's capsule. At later stages of the disease, amyloid deposits and disintegration of epithelial cells are noted in the tubules, with proliferation of connective tissue and subsequent atrophy, hyaline degeneration of the glomeruli. As a result of this, the kidneys diminish in size, presenting a picture of nephrotic contraction.

Clinical picture. The disease is insidious in onset. At first, the patient complains of debility, a reduced capacity for work, chills, loss of appetite, and intense thirst. These symptoms are followed by the appearance of pastiness and paleness of the facial skin. Oedema gradually grows to a considerable extent, resulting in facial and torso deformities. Its development is associated with a decrease in the colloid-osmotic pressure probably due to hypoproteinaemia. Oedema develops very early and rapidly and persists for months and years. It is very soft to the touch, so a slight finger pressure leaves a deep depression. Sometimes a collection of fluid is noted in the serous cavities: the abdominal, the pleural, and less often, the pericardial. In some cases, the amount of accumulated fluid is as large as 20 kg and more. Oedema at first develops in the eyelids, the abdomen, the small of the back, and the genitalia. At the sites of largest collection of fluid the skin is lustrous from tension and is sometimes ruptured, with ensuing effusion of fluid and subsequent infection, erysipelas in particular. The oedematous fluid is deficient in proteins (0.1-0.3 per cent) and rich in salts, particularly chlorides. The level of lipoproteins is elevated; therefore, the fluid acquires a milky consistency. The cardiovascular system exhibits no particular changes, if nephrosis is unaccompanied by oedema in the serous cavities. The cardiac sounds are slightly muffled. There is bradycardia, hypotension, and sometimes a liver enlargement. Considerable changes are noted in the urine and blood. The quantity of urine is reduced to 300-400 ml, its specific gravity is very high, reaching 1.040-1.060 and larger figures. A characteristic sign of lipoid nephrosis is substantial proteinuria which may rise as high as 100-120 pro mille but more often varies from 10 to 20 pro mille. The protein loss in 24 hours is sometimes as large as 10-15 g. Proteinuria is due to an increased permeability of the glomeruli and reduction in the reabsorption of protein in the tubules as well as to an easy filtration of protein possibly caused by a change in its properties. It is supposed that protein is also secreted by the tubular epithelium.

The urine excreted contains all fractions of protein, mainly albumins, and a smaller amount of globulins. An important diagnostic sign is detection in the urinary sediment of birefractive substances, cholesterol esters, which are discovered by means of polarizing microscopy. Lipoids in the urine were for the first time discovered by Munk in 1913, who gave this disease its name of lipoid nephrosis. The urinary sediment, in addition to lipoids, contains a considerable number of epithelial cells, leucocytes, and hyaline and epithelial casts. No erythrocytes are found in the urine.

A characteristic feature of nephrosis is marked hypoproteinaemia, a reduction in the protein level from the normal 7-8 g/100 ml or to 3-5 g/100 ml. In addition to hypoproteinaemia, dysproteinaemia is also present, i.e., a change in the albumin-globulin coefficient, which declines to 1 and lower, with a corresponding increase in the globulin content of the blood, mainly α_2- and β-globulins, and with a reduction in the level of γ-globulins. Hypoproteinaemia and dysproteinaemia become the cause of oedema due to a decrease in the osmotic pressure of proteins. Fluid fails to be retained in the blood and infiltrates the tissues. The quantity of chlorides excreted with the urine per 24 hours is reduced from 10-15 g to 1 g, which is accounted for not only by their retention in fluid within tissues but also by an increased secretion of aldosterone, an adrenocortical hormone which intensifies reabsorption of sodium ions in the tubules.

An increase in the levels of fat (hyperlipaemia) and cholesterol in the blood serum is also a sign of lipoid nephrosis. The cholesterol content is found to be considerably increased, up to 300-400 mg/100 ml, and sometimes even higher. The amount of lecithin and fatty acids also grows. Other symptoms are hypochromic anaemia, lymphocytosis, eosinophilia, and an elevated ESR. The nonprotein nitrogen content of the blood is usually within normal limits. Only at later stages, in case of kidney contraction, are nitrogenous substances retained and uraemia sets in. The glomerular filtration and tubular reabsorption are usually intensified (the latter, up to 99 per cent).

Lipoid nephrosis is a chronic disease, which may last for 2 years and longer. A reduction in the γ-globulin content of the blood plasma and the associated disturbance in the formation of antibodies, as well as oedema of tissues are responsible for a diminished resistance of the body to infection. In this connection, various complications ensue, such as pneumonia, pneumococcal peritonitis, erysipelas, etc. At present, these complications readily respond to antibiotics and sulphonamides. A combination of lipoid nephrosis with amyloidosis or an oedematous-albuminuric form of chronic nephritis may give rise to insufficiency of renal function resulting in uraemia.

Differential diagnosis. The possibility of lipoid nephrosis occurring in true form is denied by many contemporary authors. Others consider it a rare disease, particularly in adult persons. In the overwhelming majority of cases, lipoid nephrosis is associated with chronic nephritis or amyloidosis. Lipoid nephrosis in true form should be differentiated from the oedematous-proteinuric form of chronic nephritis and amyloid-lipoid nephrosis. The oedematous-proteinuric form of chronic nephritis is characterized by microhaematuria, moderate hypertension and a gradually developing uraemia. In amyloidosis of the kidneys the patient usually has a history of suppurations, syphilis, and tuberculosis. At the same time, not only the kidneys but also other organs (liver, spleen, intestine) are affected, while hypercholesterolaemia is unmarked. In amyloidosis, just as in chronic nephritis, there is moderate renal insufficiency and hypertension, a positive Bennhold's test with an intravenous infusion of 10 ml of a 1 per cent Congo red solution, which is retained by amyloid.

Prognosis. During the past few years, prognosis has become much more hopeful owing to the application of antibiotics, chemopreparations and hormones. A recovery may be considered when the protein composition of the blood and the ESR have returned to normal, proteinuria has ceased and oedema subsided.

The *prophylaxis* of lipoid nephrosis consists in a vigorous treatment of diseases likely to cause this condition, elimination of infectious foci in the body, hardening of the body by engaging in sports, physical culture, etc.

Treatment. This consists of radical measures against the principal disease responsible for lipoid nephrosis, as well as infectious foci, using antibiotic therapy. Surgical intervention is also necessary sometimes (for suppurative processes). Oedema is reduced by means of bed rest, restriction of water intake to 500-600 ml in 24 hours and common salt to 2-3 g per day. Diuresus is promoted by the administration of potassium chloride in a dose of 5 g in 24 hours, a diet rich in potassium salts (potatoes, fruit, etc.), and diuretics (novurit, phonurit, hypothiaside, etc.). The diet must be sufficient in caloric value and contain 120-140 g of protein, 350-400 g of carbohydrates, and 30-40 g of fats. The protein intake is considerably limited only in case of renal insufficiency (amyloid kidney contraction). Thyroidin is also prescribed in a dose of up to 1 g in 24 hours. In case of tachycardia and pains in the heart region, thyroidin therapy should be discontinued. Of late it has been recommended to administer ACTH (20-30 units twice daily), prednisone, and prednisolone (in doses of 5-10 mg 2-3 times daily).

This disease is treated successfully at climatic resorts where low humidity and high temperature of the air as well as a still weather contribute to dilation of vessels and intensified perspiration. Climatotherapy is recommended for a period of 2-3 months.

Amyloid-Lipoid Nephrosis (nephrosis amyloidea)

Amyloidosis is a protein dystrophy of tissue elements with the formation of a dense homogenous substance which Virchow named amyloid. He erroneously presumed that in respect of its properties amyloid was close to carbohydrates of animal origin (from Greek *amylon*—starch). Later, N. Kravkov proved that for its properties amyloid is close to proteins and is a complex combination of protein with chondroitin sulphuric acid.

Aetiology and pathogenesis. Amyloidosis is secondary to a number of pathologic conditions of the body, such as tuberculosis, particularly that in the bones and joints, osteomyelitis, chronic pulmonary suppurations (bronchiectasis, pleural empyema, abscess, actinomycosis, etc.), rheumatoid arthritis, systemic lupus erythematosus, chronic leucosis, and myelomic disease. In rheumatoid arthritis, amyloidosis affects not only the kidneys but also the spleen, adrenal glands, liver, etc. An electrophoretic examination of the protein fractions of both the blood serum and urine shows the prevalence of α_2- and γ-globulins. The incidence of amyloid-lipoid nephrosis among men is two times that among women. Cases of this disease in children are quite rare.

Pathologic anatomy. An amyloid-lipoid kidney is enlarged, dense in consistency, with a waxy hue visible on section (so-called enlarged waxy kidney). In case of contraction of the kidney, which is noted at terminal stages, the kidney becomes smaller in size, its surface is rough, and the capsule peels off with difficulty (so-called amyloid contracted kidney). Microscopy reveals amyloid deposits under the endothelium of the glomerular capillaries. The capillaries are gradually obstructed by amyloid masses, and the blood flow in the glomeruli stops. The tubules undergo degenerative changes and lipoid infiltration. Amyloid-lipoid nephrosis is, as a rule, attended with amyloidosis of the spleen, liver, intestine, adrenal glands, lymphatic glands, pancreas, thyroid gland, etc.

Clinical picture. The classical symptoms of amyloid lipoid nephrosis are oedema, proteinuria, hypercholesterolaemia, and, at a later stage, azotaemia. The above-mentioned symptoms are not simultaneous in onset but develop at different periods of the disease. In view of this, it is subdivided in clinical practice into the following three developmental phases: (a) proteinuric; (b) oedematous-hypotensive; and (c) azotaemic. As a rule, amyloidosis at first develops in the spleen and later in the kidneys, with the onset of albuminuria. In this period, the clinical symptoms of the principal disease responsible for a kidney lesion prevail. Proteinuria in combination with enlargement of the spleen is the initial symptom of amyloid-lipoid dystrophy. Later, amyloidosis extends to the liver and the adrenal glands, with the development of protein deficiency and adrenal hypofunction (oedematous-hypotensive phase). Subsequently, symptoms of renal insufficiency (azotaemic phase) make their appearance.

The proteinuric phase. The principal sign of this phase is proteinuria. At first, only traces or a small quantity of protein are detected, but later its amount increases with the progression of the disease. The urine protein contains much globulin, particularly β- and γ-globulins and fibrinogen. The second most frequent sign is mild haematuria, which follows in the wake of proteinuria. The urinary sediment may also contain leucocytes, and granular and waxy casts, if amyloidosis is joined by a lipoid affection of the kidney. In the proteinuric phase, glomerular filtration is elevated, reabsorption is diminished, and the total amount of urine discharged is increased; there is nycturia (nocturnal urinary incontinence), but no marked hypoproteinaemia, and no hypercholesterolaemia. The cardiovascular system shows no substantial abnormalities. The arterial pressure is, as a rule, within normal limits or is slightly elevated, while no changes are found in the fundus oculi. No disorders are detected in digestive function. The red blood count is within normal limits, with neutrophilic leucocytosis and an elevated ESR due to the prevalence of globulins in the blood. The test with Congo red is negative because of an insignificant deposition of amyloid in organs. The albuminuric phase may last from three months to five years, depending on the principal disease responsible for amyloidosis. The duration of the proteinuric phase is longer in suppurative processes and shorter in tuberculosis.

The oedematous-hypotensive phase. In this phase, the symptoms of the principal disease are relegated to the background and oedema makes it appearance. The face becomes pale and puffy. The skin is dry and comes off in scales. The cardinal symptom of this phase is oedema which commonly develops gradually and sometimes acutely within 24 hours in connection with exacerbation of the principal disease or a new infection (pneumonia, influenza, erysipelas, etc.). Other factors likely to contribute to the development of oedema are chilling, diarrhoea and blood loss. Oedema at first appears in the feet and shanks, and later in the thighs, the small of the back and the genitalia. In far-advanced cases, oedema also develops in the serous cavities (pleural, peritoneal, pericardial).

Another symptom of this phase is hypotension, the systolic pressure dropping to 100 mm Hg. Hypotension is apparently associated with adrenal hypofunction. In this condition, the patient complains of dizziness. Proteinuria progresses, often growing as high as 10 pro mille and more, with prevalence of globulins in the urine. The urinary sediment contains leucocytes, renal epithelial cells, and hyaline, granular, epithelial and waxy casts. In addition, the presence of a large number of cholesterol ester crystals detectable by polarizing microscopy is evidence of amyloidosis having been joined by dystrophic changes in the tubular apparatus, i.e., lipoid nephrosis. Renal function is considerably disturbed, the amount of urine is diminished, and there is nycturia and a reduction in the filtrative and an increase in the reabsorptive capacity of the kidneys. A reduction in filtration is partly due to a fall of the arterial pressure and a diffuse amyloid degeneration of the glomeruli, while an increased reabsorption is caused by the concomitant lipoid nephrosis joining amyloidosis. The fundus oculi is either normal or shows only a slight change. The total protein content of the blood is reduced to 4 g/100 ml, whereas the cholesterol level is increased to 800 mg/100 ml and more. The heart is moderately enlarged at the expense of the left ventricle, the cardiac sounds are muffled, with a systolic murmur audible over the heart apex. There is a disturbance in digestive function: a loss of appetite, gastric and intestinal dyspepsia, and reduction in the secretory function of the stomach, even to the degree of gastric achylia. Hepatic function is deranged in respect of protein and carbohydrate metabolism. The ESR is elevated, with progression of neutrophilic leucocytosis, and the development of moderate anaemia. The albumino-hypotensive stage does not last longer than one year.

The azotaemic phase. The most important symptom of azotaemic uraemia is renal insufficiency resulting from contraction of the kidneys (contracted amyloid kidney). At the same time, symptoms of dyspepsia become much more pronounced, as manifested by nausea, vomiting, and diarrhoea. Changes in the central nervous system are less pronounced than in other forms of renal insufficiency: the level of nonprotein nitrogen is rarely higher than 100 mg/100 ml, while the arterial pressure is within normal limits or slightly elevated. At the same time, there is a notable reduction in the concentrating function of the kidneys, a diminution of the filtration and reabsorption capacity, hypo- and isosthenuria, displacement of the renal epithelium by scar tissue, abatement of oedema, persistent hypercholesterolaemia and hypoproteinaemia, and increased anaemia. Amyloid-lipoid nephrosis is frequently accompanied by complications. One of them is thrombosis of the renal veins consequent upon accumulation of globulins and fibrinogen in the glomeruli and deceleration of the blood flow. This is attended with pains in the small of the back, a rapid decrease in the amount of urine discharged, an increase in haematuria and an acute development of hypertension and azotaemia. Another complication is purulent peritonitis.

Differential diagnosis. Differential diagnosis is facilitated by Bennhold's test with an intravenous infusion of 10 ml of a 1 per cent Congo red solution. The stain is retained by an amyloid kidney, liver, spleen, intestine, etc., as a result of which not more than 50 per cent of it is left in the blood one hour after its injection. To ascertain the degree of stain absorption by amyloid organs two blood specimens are taken from another vein, one four minutes and the other one hour after the dye had been introduced. The colours of both specimens are compared in a colorimeter. Amyloid-lipoid nephrosis must be differentiated from lipoid nephrosis, the oedematous-proteinuric form of chronic nephritis, primary and secondary contraction of the kidney, and hypertensive vascular disease. In pronounced hypertension, the course of amyloid-lipoid nephrosis resembles that of chronic nephritis. However, predominant localization of oedema in the lower extremities, enlargement of the liver and spleen, and relatively unmarked haematuria are evidence in favour of amyloid-lipoid nephrosis.

The *prognosis* is serious and depends, on the one hand, on the course of the principal disease responsible for amyloid-lipoid nephrosis and the possibility of radical recovery, and, on the other hand, on the degree of advancement of amyloidosis. In the presence of amyloid contraction of the kidney, the prognosis is ominous: the patient dies of azotaemic uraemia or various complications (pneumonia, peritonitis, etc.).

Prophylaxis and treatment. Prophylaxis consists in a timely and radical treatment of all diseases likely to cause amyloidosis. Therapeutic measures must be complex and directed to eliminating both amyloidosis and the major causes of amyloid-lipoid nephrosis. In nephrosis of tuberculous aetiology specific therapy with phthivazide, streptomycin, PASA and other drugs is prescribed, while in the case of syphilitic aetiology antisyphilitic therapy is required. For suppurative processes in the lungs antibiotics are administered in large doses. In addition to intramuscular injections, intrabronchial, and for empyema, intrapleural injections are desirable. Should this treatment prove ineffective, surgery is required, in particular for osteomyelitis, tuberculosis in the bones and joints, pulmonary tuberculosis, and pulmonary suppurations. In symptomatic treatment, great importance is attached to dietotherapy. In cases of marked oedema and hypoproteinaemia, the diet includes a large quantity of animal fats: 1.5-2 g per kg of body weight. Intravenous infusions of whole blood and plasma and subcutaneous injections of various protein hydrolysates are recommended. In azotaemia, the protein intake is considerably restricted in favour of carbohydrates. In hypercholesterolaemia, a diet low in fats is prescribed. In the presence of extensive oedema the daily intake of sodium chloride should not exceed 2-3 g. The liquid intake is also restricted. Various diuretics such as novurit, hypothiaside, and phonurit are indicated for this disease. It is also recommended to undertake hormone therapy with thyroidin, prednisone, prednisolone, etc. In the azotaemic phase, the patient is kept on an exclusive sugar and fruit diet on periodically appointed days. In this phase, liquid intake is not restricted. In case of hypochloraemia, 10 per cent sodium chloride is injected intravenously in a dose of 10-20 ml. Acidosis is treated by the administration of alkaline water per os and through an enema.

Necrotizing Nephrosis (nephrosis necrotica)

Necrotizing nephrosis develops in the epithelium of the convoluted tubules and causes a diminution in the urinary secretion and acute renal insufficiency.

Aetiology and pathogenesis. The principal cause of acute necrotizing nephrosis is poisoning with salts of heavy metals (mercury, bismuth, uranium, chromium, gold, lead, etc.), strong organic and inorganic acids (sulphuric, hydrochloric, phosphoric, oxalic, etc.), barbiturates, sulphonamides, potassium chlorate, carbon tetrachloride as well as with antifreezing agent widely used in motor engines. Of the infectious diseases liable to produce necrotizing nephrosis mention is deserved by cholera, dysentery, diphtheria, leptospirosis, as well as puerperal and postabortion sepsis. The disease is also frequently observed after transfusion of incompatible blood and following intestinal obstruction. Necrosis of the tubular epithelium is due to the direct action of toxic substances causing tissue coagulation. Selective affection of the tubules is accounted for by the latter's being capable of absorption as a result of which the concentration of toxic substance within them attains a maximum. Oliguria or anuria is accounted for by a disturbance in the renal blood flow as well as by obstruction of the glomerular lumina by desquamated epithelial cells and decomposition products of haemolysis. In the period of convalescence polyuria is due to a disturbance of absorption in the tubules since the newly regenerated epithelium of the convoluted tubules has not yet developed an adequate capacity for reabsorption. Acute renal insufficiency progresses along with a diminution of the urinary secretion.

Pathologic anatomy. The kidneys are enlarged, soft in consistency and flaccid and the capsule peels off readily. Histological investigation reveals necrosis of the tubular epithelium, a pink-coloured granular mass filling the lumina of the tubules. The glomerular capillaries are sometimes congested. After the 3rd or 4th day of the disease, regenerative processes set in and the tubular structure is fully repaired after a certain period. In extremely rare cases, the process ends in kidney contraction.

Clinical picture. The urinary secretion is diminished from the very first day of the disease. In mild cases, the urinary secretion is not disturbed substantially, and in moderately grave cases, the amount of urine does not exceed 500 ml, while in severe cases total anuria sets in. Anuria lasting for 5 to 7 days is an unfavourable sign. The urine resembles soapsuds in colour and contains a small quantity of protein. Microscopy of the urinary sediment reveals renal epithelial cells which have undergone fatty degeneration, isolated hyaline and granular casts, microhaematuria and leucocyturia. In rare cases, there is an increase in the arterial pressure. Necrotizing nephrosis may give rise to renal insufficiency manifested by nausea, vomiting, and diarrhoea. These are later joined by pericarditis, pleuritis and Kussmaul's laboured respiration, and coma. Anaemia, neutrophilic leucocytosis, and azotaemia are also encountered.

Recovery is manifested by polyuria. The urinary secretion grows up to 3 litres in 24 hours. The urine is of a pale colour, a low specific gravity, and with a slight proteinuria. Soon, the amount of urine returns to normal, and azotaemia abates. Both the red and the white cell counts return to normal along with a reduction in the nonprotein nitrogen level and a deceleration of the ESR. All symptoms of acute necrotizing nephrosis disappear. The polyuric period lasts for 2-3 weeks, a disturbance in the renal concentration sometimes persisting for months. In severe cases, the accumulated products of protein decomposition fail to be eliminated from the body in spite of the presence of polyuria, and the patient dies of uraemia. The clinical picture of necrotizing nephrosis has certain specific features depending on the cause of poisoning.

Sublimate Necrotizing Nephrosis

Soon after the administration of corrosive sublimate in solution or tablet form, the patient senses a metallic aftertaste in the mouth; a burning sensation and pain in the mouth, along the course of the oesophagus and in the stomach, and diarrhoea with a discharge of blood and mucus appear. The oral mucosa becomes affected with ulcerative stomatitis. In 2-3 days' time, and in case of large-dose poisoning even earlier, intense pains are felt in the small of the back, the urinary secretion diminishing to the degree of anuria. This grave condition is caused by necrosis of the renal tubules, the urine being found to contain a considerable amount of protein (within range of 5-6 pro mille, sometimes more than 10 pro mille), the urinary sediment containing hyaline, granular and epithelial casts, and erythrocytes. The specific gravity of the urine is at first high, and later begins to diminish when the insufficiency of nitrogen excretion by the kidneys aggravates and azotaemic uraemia makes its appearance. There is headache, nausea, vomiting, an arterial pressure increase, and progressive anaemia. The amount of nitrogenous wastes in the blood and tissues increases, and the patient dies in a state of coma.

The diagnosis rests on the past history of a case (evidence of administration of corrosive sublimate). In doubtful cases, it is necessary to examine the urine or blood for the presence of mercury.

Post-Transfusion Necrotizing Nephrosis

Transfusion of incompatible blood causes affection of the epithelial lining of the tubules and their obstruction by decomposed haemoglobin. The patient complains of pains in the small of the back and breathlessness. There is pallor of the face and cold sweats, frequent vomiting, and a disturbance in the urinary secretion, even to the degree of anuria. The urine is dark in colour due to the presence of a considerable amount of haemoglobin (haemoglobinuria), with much protein and many casts. The spleen and liver are enlarged, and haemolytic jaundice develops. If the disease runs an unfavourable course, anuria and azotaemia will ensue. In other respects, the progress of the disease is the same as in sublimate necrotizing nephrosis.

Similar changes may occur in the body in case of poisoning with strong inorganic and organic acids, symptoms of gastrointestinal involvement appearing simultaneously (oesophagitis, gastritis, haemorrhagic colitis). In poisoning with antifreezing agent the liver is involved in addition to the kidneys. In poisoning with narcotic drugs necrotizing nephrosis is attended with an intense excitement followed by a deep sleep.

Treatment. Poisoning is treated by means of gastric lavage, saline purgatives, siphon enemas, the patient being given much water to drink (see "Poisoning"). In the case of sublimate poisoning, a special antidote, antidotum metallorum (hydrogen sulphide solution), is introduced into the patient's stomach in order to convert mercury bichloride into harmless sulphide of mercury. Ten minutes after the administration of the antidote, lavage of the stomach is undertaken. White of egg whipped in water is given per os, as well as several glassfuls of milk. The latter causes mercury to settle in the form of insoluble albuminate. A 5 ml dose of a 5 per cent unithiole solution is injected subcutaneously or intramuscularly once in every six hours, and in the subsequent 6-7 days, two or three times in 24 hours. Because of the likelihood of azotaemia, the daily intake of protein is limited to 25 g at the expense of an increase in carbohydrates and fats. The intake of liquid must not be larger than 700 ml, with a restriction on common salt. In the event of persistent vomiting, 10 ml of a 10 per cent common salt solution is infused intravenously. A disturbed renal blood flow is restored by the application of UHF currents and diathermy to the lumbar region. Perirenal novocain blocking is performed. For control of cardiovascular insufficiency, it is recommended to use an intravenous infusion of 20-40 ml of a 40 per cent glucose solution with strophanthin, or a subcutaneous injection of 500 ml of a 5 per cent glucose solution, as well as a 1 per cent solution of mesaton or sympatol, and cordiamine, caffeine, camphor. Another remedy is blood-letting followed by transfusion of donor blood.

During the past few years, acute necrotizing nephrosis has been treated by haemodialysis with the aid of an artificial kidney apparatus, particularly in cases of poisoning with mercury, arsenic, copper, mushrooms, barbiturates, etc. This type of therapy is contraindicated for gastrointestinal haemorrhage and hypertension.

Nephropathy of Pregnancy (nephropathia gravidarum)

Toxaemia of pregnancy develops in the latter half of pregnancy, and sometimes after labour, and is characterized by oedema, albuminuria, hypoproteinaemia, hypertension, and in grave cases, by changes in the fundus oculi and eclampsia. In view of the presence of symptoms characteristic of a kidney disease many call this condition nephropathy of pregnancy. Of late, there has been a considerable decline in the incidence of this disease.

Aetiology and pathogenesis. The origin of nephropathy of pregnancy is attributed to infection, often by streptococci, and to a deficit of various substances of protein origin in the body of a pregnant woman. A number of authors trace the origin of nephropathy to a precursory kidney disease or to a circulatory disorder in the kidney and hypersecretion of rennin. It is also regarded as a manifestation of allergy, and hyperfunction of the endocrine glands: the thyroid and the pituitary body. Among the contributing factors may be unfavourable climate, nutrition, and working and living conditions, as well as nervous and emotional shock. The role of chilling is apparently unsubstantial. The mechanism of the development of hypertension in nephropathy of pregnancy is the same as that seen in hypertensive vascular disease. In nephropathy of pregnancy, however, hypertension disappears after labour without leaving a trace. The development of oedema is associated with hypoproteinaemia and reduction in the colloid-osmotic pressure, an intensified formation of the antidiuretic hormone in the pituitary body and aldosterone in the adrenal glands. The latter retains sodium ions in tissues. The oedematous fluid is deficient in protein (0.1-0.3 per cent). Considerable changes in the nervous and endocrine systems are accompanied by spasm of the smaller vessels and capillaries, especially in the brain. Spasm of the renal vessels causes a nutritional disturbance in the glomeruli. Spasm of the brain vessels leads to cerebral oedema, which plays a leading role in the development of nephropathy of pregnancy and convulsions. Changes in the urine are the same as those seen in the oedematous-proteinuric form of chronic nephritis.

Pathologic anatomy. The kidneys are enlarged, the capsule peels off readily, and the cortical layer is dull in colour and lusterless. Microscopy reveals degenerative changes in the glomeruli, changes in the tubules resembling those in nephrosis, though lipoid infiltration of the tubules is less pronounced. The glomeruli are enlarged and exsanguinated (bloodless), which is characteristic of nephropathy of pregnancy; haemorrhages and foci of necrosis are noted in the kidneys (cortical layer), brain, liver, and spleen.

Clinical picture. Nephropathy of pregnancy is known to occur in two varieties: (a) oedematous or protein-oedematous, and (b) hypertensive. The patient complains of headache, dizziness, dyspnoea, dyspeptic symptoms, thurst, pains in the small of the back, frequent urination, impairment of vision, and a sensation of specks before the eyes. Oedema begins to develop in the lower extremities and gradually invades the thighs, abdomen, small of the back, neck, and less frequently the face. Oedema of the serous cavities is a relatively rare occurrence.

Objective examination, in addition to oedema, reveals displacement of the left heart boundaries, muffled cardiac sounds, bradycardia; in severe cases, tachycardia, and gallop rhythm. Signs of cardiac insufficiency appear sometimes, such as cardiac asthma, blood-spitting, and enlargement of the liver. The systolic arterial pressure is at first within a range of 170-180 mm Hg, the diastolic, 90-100 mm Hg. During attacks of eclampsia, the pressure is considerably elevated (up to 200/120 mm Hg and higher). The respiratory organs show no particular abnormalities. Examination of the fundus oculi reveals symptoms of angiospastic retinitis: constriction and tortuousness of arteries and dilatation of veins, haemorrhages in the fundus oculi, oedema and degenerative changes in the retina, and sometimes atrophy of the optic nerve. An obvious affection of the fundus oculi is an indication for abortion. There are also pains in the hepatic region and liver enlargement. The urinary secretion does not exceed 500 ml in 24 hours, the specific gravity of the urine is high, the chloride content of the urine is diminished and that of protein elevated to 6-10 pro mille, with microhaematuria. Azotaemia is, as a rule, absent, and in considerable oliguria it is moderately elevated. A moderate hypochromic anaemia is encountered. In eclampsia of pregnancy there are a neutrophilic leucocytosis, eosinophilia and monocytosis, an elevated ESR, hypoglycaemia, hypercholesterolaemia, and hypoproteinaemia (3-5 g/100 ml) at the expense of a reduction in albumins. Eclampsia more frequently runs its course in the form of convulsions and coma. The latter form is the most severe, with a fatal outcome. Attacks of eclampsia are attended with vomiting, an elevation of the arterial pressure, a loss of vision down to transient total blindness, and convulsions lasting for several minutes. Eclamptic attacks resemble those of renal eclampsia and recur several times a day. In the periods of remission the patient's subjective condition is satisfactory.

Differential diagnosis is established between nephropathy of pregnancy and hypertensive vascular disease, chronic and acute nephritis and a number of urologic diseases. As a rule, hypertensive vascular disease and nephritides are detected in the early months of pregnancy, while nephropathy of pregnancy is diagnosed at the end of this period. Hypertensive vascular disease is accompanied by an isolated increase in the arterial pressure, without oedema and urinary syndrome. Besides that, symptoms of nephropathy disappear after the termination of pregnancy. Nephropathy of pregnancy should be differentiated from chronic nephritis by means of considering the past history of a case and detection of early symptoms of kidney affection. In addition, in chronic nephritis the specific gravity of urine is reduced and symptoms of renal insufficiency come on the scene, as distinct from nephropathy of pregnancy. Evidence in favour of acute nephritis is infection, microhaematuria, cylindruria, and dropsy of the serous cavities. Nephropathy should also be differentiated from insignificant pitting of the shanks in the late months of pregnancy, proteinuria and slight haematuria as a sign of a certain circulatory insufficiency.

Prognosis. This has become more favourable of late owing to early identification and prophylactic and therapeutic measures. If effective assistance is unavailable, intrauterine death of the foetus or death of the infant in the early days of life occurs. In the majority of cases, nephropathy of pregnancy ends in complete recovery;

in repeated pregnancy, however, the disease is liable to recur. Some women recovered from nephropathy of pregnancy may develop hypertensive vascular disease and cardiovascular insufficiency. Sometimes nephropathy of pregnancy gives rise to chronic nephritis.

Prophylaxis is carried out at maternity health centres up to the time of labour, systematic observations being made over the weight, arterial pressure, urine, and fundus oculi. Gravid women with hypertensive vascular disease, chronic nephritis, and with a history of nephropathy of pregnancy are subject to special registration. Termination of pregnancy is considered in case of detection of hypertensive vascular disease or chronic nephritis.

Treatment. Upon the appearance of symptoms of toxaemia of pregnancy the patient should be taken to hospital or confined to bed at home. A diet is prescribed with restrictions on common salt and liquids. In order to meet the requirements for proteins which are indispensable for the formation of a foetus, a diet is prescribed which contains at least 100 g of proteins (boiled meat, fish, cottage cheese, eggs, etc.), 50 g of fats, and 400-500 g of carbohydrates. In the presence of oedema an exclusive diet of fruit (1.5 kg of apples and 100 g of sugar) or sugar and cottage cheese (200 g and 500 g respectively) is prescribed on certain days.

In view of the substantial role played in the development of nephropathy of pregnancy by an increased excitability of the neuropsychic sphere it is helpful to administer sedatives (bromides. tincture of valerian, etc.), soporifics and antispastic drugs (intravenous or intramuscular injections of a 25 per cent magnesium sulphate solution in a dose of 10-20 ml, a 2 per cent dibazol solution injected twice daily in a dose of 2 ml or given per os in a dose of 0.05 g twice daily, and sometimes chloral hydrate by means of enema in a dose of 2 g per 50 ml of water). For a high-degree hypertension reserpine, serpasil, ghendon (raunatin), and other agents are administered. Mercury preparations, such as novurit, as well as hypothiaside, calcium chloride or ammonium chloride are used as diuretics. For an acute circulatory failure, intravenous infusions of strophanthin are given in a dose of 0.5 ml of a 0.05 per cent solution in 10 ml of a 40 per cent glucose solution, and cordiamine in a dose of 2 ml is administered subcutaneously 2-3 times daily. Eclampsia of pregnancy is managed by the same methods as renal eclampsia (see p. 296) Another pregnancy is contraindicated during the coming 2-3 years.

PTOSIS OF THE KIDNEYS (NEPHROPTOSIS)

Aetiology. Kidney ptosis is caused by a reduction of the intra-abdominal pressure, a diminution or disappearance of the perirenal adipose cellular tissue, and underdevelopment of the abdominal muscles or a reduction in their tone, which is found especially frequently in mothers of large families who neglected the rules of hygiene during pregnancy, and in emaciated persons. At the same time, a simultaneous ptosis of other organs of the abdominal cavity is also observed. The right kidney is more liable to ptosis than the left. Kidney ptosis should be distinguished from its congenital malposition.

Clinically, there is a difference between the movable kidney (ren mobile), when the kidney only slightly slides from its normal position, and the wandering or floating kidney (ren migrans) when the kidney is displaced and freely movable inside the adbominal cavity. Displacement of the kidney often causes prolonged excruciating pains.

Clinical picture. The first to describe ptosis of the kidney as a disease was S. Botkin.

The patient complains of dull pains in the upper part of the abdomen, often accompanied by nausea, sometimes with paroxysms of vomiting, and shooting pains in the lumbar region radiating to the thigh, the rectum and the external genitalia. Reflected pains resembling angina pectoris are sometimes felt in the heart region. The intestine is most commonly prone to constipation, less often to diarrhoea. Pain mitigates at night during sleep and rest, i.e., when a person is lying down and, on the contrary, grows worse during walking, physical exertion, and jolty riding, which is accounted for by a large displacement of the kidney. Botkin pointed out in his time that these prolonged incessant pains may drive the sufferer to hysteria.

A considerable displacement of the kidneys may result in bending and twisting of the ureter with complete closure of its lumen, which entails urinary retention of variable degree, distention of the kidney pelvis (hydronephrosis) and facilitates penetration of infection from the intestine into the kidney pelvis followed by the latter's inflammation. Bending or twisting is accompanied by intense pains in the small of the back, which radiate to a leg, a groin, and the genitalia. The diagnosis of kidney ptosis is made easier by the kidney, usually unpalpable in healthy persons, becoming accessible to palpation in case of a ptosis. Palpation should be carried out both in an upright and recumbent position of the patient, as was pointed out by Botkin in his time.

In ptosis of the kidney, a small quantity of protein and erythrocytes sometimes appear in the urine, but the function of the organ remains undisturbed.

Treatment. A highly nourishing diet is prescribed, which gives a considerable effect in mild kidney ptosis. This diet in combination with hygienic exercises aimed at strengthening the adbominal muscles proves a success in many cases. General invigorating therapy is also indicated. It is recommended to wear an abdominal supporting bandage. Strong tightening of the abdomen with belts or laces is forbidden. Only in severe cases is surgery required, which consists in suturing the floating kidney to the omentum.

TUMOURS OF THE KIDNEYS AND THE URINARY PASSAGES

Tumours of the kidneys are encountered much less frequently than tumours of other organs. The most frequent kidney tumour is hypernephroma taking its origin from the wandering cells of the adrenal gland which penetrated the renal tissue. Cancer and sarcoma are observed very rarely, and benign tumours are even less frequent. Villous polyps, papillomas, and carcinoma may be encountered in the urinary bladder.

Hypernephroma is very liable to metastasize, particularly to the bones, lungs, liver and lymphatic glands.

Clinical picture. The signs of a kidney or urinary bladder neoplasm are as follows: a tumour, haematuria with proteinuria, and at later stages, cachexia with marked anaemia. The most important of them is haematuria, which is often the only symptom of a tumour. Haematuria sometimes occurs in perfectly healthy persons, may be of short duration, and may not reappear for several months; in other cases, haematuria is discovered only by microscopy or may be massive and prolonged, causing severe anaemia and extreme emaciation. The source of haemorrhage is detected by means of a test with three glasses (Fig. 49).

The blood in the urine appears as a result of haemorrhage from the kidney, the urinary bladder or urethra. In order to locate the site of bleeding the above-mentioned test is used. The patient is told to void urine at first into the first glass, then into a second, and a third. If blood is detected in all three glasses the bleeding is from the kidney; the presence of blood only in the third glass is evidence of haemorrhage from the urinary bladder; blood discovered only in the first glass is effused from the urethra.

In the diagnosis of a tumour of the kidney or the urinary bladder an important role is played by cystoscopy and pyelography.

In the late stages of the disease, dull and nagging pains appear in the lumbar region. In case of tumour pressure on the roots of the adjacent nerves, shooting girdle pains are felt. Pains appear also at the sites of metastases: in the bones, liver and lungs.

Palpation usually reveals neoplasms in the lower pole of the kidney, where a dense, knobby and nonmovable tumour is felt with the fingers. Sometimes it grows to a considerable size, extends as far as the lower surface of the liver and spleen, and raises the roof of the diaphragm on the dependent side displacing the adjacent organs to the side and upwards. Tumours in the upper pole of the kidney are inaccessible to palpation.

Cachexia develops in prolonged haemorrhage and with far-advanced tumours. The duration of the course of tumour growth is variable. In cases not treated surgically survival from the time when the diagnosis is made is about 2-3 years.

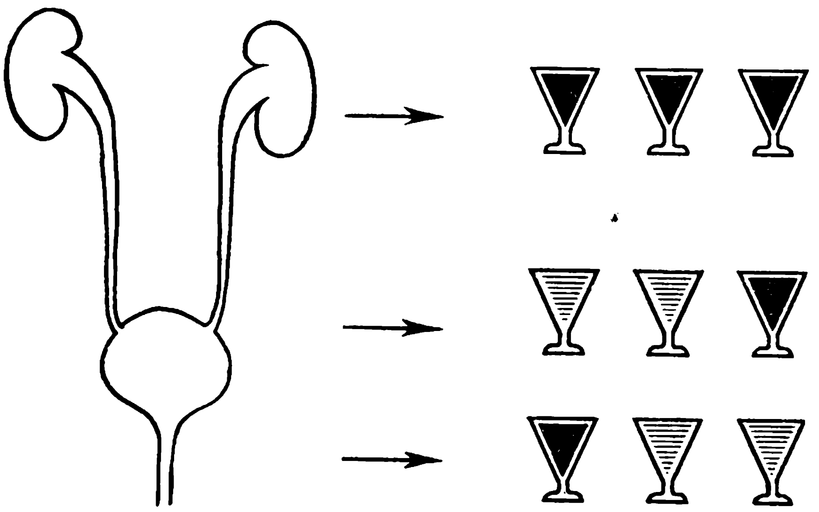

Fig. 49. Test with three glasses

Death occurs at a relatively early date in case of metastases which are localized mostly in the liver, lungs, and bones, above all in the vertebrae, thigh and skull.

Treatment. Radical surgery on the affected organ may bring about complete recovery. A symptomatic remedy for intense pains are narcotics, such as pantopon and morphine.

NEPHROLITHIASIS

Nephrolithiasis is a widespread disease. It develops mainly at an age of 20 to 40 years, more often in men than in women. Renal stones (calculi) form in the pelvis and calyces of one or both kidneys simultaneously. Stones may be isolated, sometimes 2-3 in number, or multiple. They vary in size from a grain to a child's head and in weight from fractions of a gram to 2 kg. Stones of not more than 1 cm in size may pass into the ureter, sometimes obstructing its lumen over a length of 10-15 cm. The shape of the stones depends on the site of their location: in the renal pelves they are oval or acquire a peculiar form—the so-called coral calculi, in the ureters they are elongated or cone-shaped. They may be faceted when they rub each other. On cross section the stones show a concentric laminar structure. The chemical composition of renal calculi is variable: in an acid medium they are urate, consisting of uric acid crystals, or oxalate, formed from potassium oxalate; in alkaline urine they are phosphate, formed from tripelphosphates. The stones may be of a mixed composition; in rare instances, they consist of cystine and cholesterol, while a prolonged therapy with sulpha drugs may cause the formation of sulphonamide calculi. Uric acid (urate) stones are of a yellow colour and hard consistency, with a smooth or granulated surface. The phosphates are of a greyish-white colour, with a rough surface and of a loose, crumbly consistency. The oxalates are very hard, with a knobby surface. They readily damage the mucous membrane and are stained black with the blood pigment.

Cystine stones are colourless, of a dense consistency; xanthine stones are of a reddish colour, with a smooth surface; cholesterol stones are black and brittle.

Aetiology and pathogenesis. The basic cause of nephrolithiasis is a metabolic disorder with a simultaneous disturbance in the condition of the urinary passages and the character of nutrition.The urine of a healthy person contains special colloidal substances, various crystalloids (uric, oxalic and other acids). Deposition of these crystalloids takes place with a change in the acid-alkaline balance, mainly in the direction of acidosis. In the formation of calculi a certain role is played by the character of drinking water; in particular, hard water rich in calcium salts predisposes to stone formation. The causes of stone formation are as follows: (1) an increased desquamation of the mucosal epithelium of the urinary passages due to inadequate supply of vitamins A, B and D; (2) retention of urine in the renal pelvis and its infection; (3) limited intake of liquid and an increased content in the urine of urates, oxalates, etc.

Pathologic anatomy. The mucous membrane of the renal pelvis, calyces and the renal parenchyma itself suffer a mechanical damage from the calculi. The extent of this damage depends on the size and shape of the stones, duration of the disease, the state of the intrapelvic pressure, and concurrent infection which is present nearly always. Urine drainage is obstructed completely or partially. Complete obstruction is caused by small stones which readily stop up the ureter. This results in total atrophy of the renal parenchyma due to an elevated intrarenal pressure, spasm of the renal vessels, and anaemization of the kidneys. Retention of urine in the pelvis, the latter's distention, and hydronephrosis occur, with infection joining urinary retention and producing pyonephrosis. Simultaneously, purulent foci arise in the kidney or the latter undergoes purulent softening. A suppurative process in the kidney may be complicated by inflammation of the perirenal connective tissue—paranephritis.

Clinical picture. The disease sometimes runs a symptomless course, the renal calculi being discovered by accident during an x-ray examination of the kidneys. Most commonly, however, nephrolithiasis is accompanied by dull or acute pains in the small of the back. Renal colic usually occurs in walking and physical strain, also in jolty riding. It is associated mainly with a small stone being jammed in the ureter, which leads to the latter's obstruction, complete cessation of urine drainage, an acute distention of the renal pelvis, and a sharp elevation of the intrarenal pressure. During renal colic pains are felt in the small of the back, its left or right halves, depending on the localization of calculi. They radiate to the testis, the genitalia, the groin, and the thigh on the dependent side and only in rare instances, to the opposite side. During a colic the patient is restless, changes his posture in an effort to lessen the pain, and cannot help groaning and crying; sometimes he suffers a collapse due to excruciating pains. The attack is attended with nausea, vomiting, retention of the stool and intestinal gases, and frequent and sometimes painful urination. The face is hyperaemic, the abdomen is inflated, and Pasternatsky's sign is positive. Sometimes, there is fever and leucocytosis, which disappear with the cessation of an attack. There may also be atypical attacks of renal colic with pains radiating to the epigastrium, the shoulder-blade, arm, sacrum and anus.

Persistent or transient dull lumbar pains associated with renal calculi occur in the presence of slightly movable stones, especially coral ones. An important sign of nephrolithiasis is micro- or macrohaematuria. It is produced by an injury to the mucous membrane of the renal pelvis or ureter in walking or physical overstrain, and is discovered after an attack of renal colic. Pyuria is secondary to infection of the urinary passages, leucocytes appearing in the urine with ensuing pyelitis or pyelonephritis. The latter runs its course with a severe chill, high-grade fever, and contributes to the destruction of the renal parenchyma and the formation of new calculi.

Nephrolithiasis is sometimes accompanied by anuria resulting from obstruction of the ureters by bilateral calculi. Anuria is a dangerous symptom since it is shortly followed by azotaemic uraemia and death occurring within a matter of 10-14 days.

In typical cases nephrolithiasis is easily recognized. In addition to the above-mentioned symptoms, certain tests are also important for a diagnosis. They are as follows: (a) examination of urine for the content of erythrocytes before and after walking; an increase in the number of erythrocytes after walking or physical exertion is evidence in favour of the presence of renal calculi; (b) the test with indigo carmine, which flows out with a delay from the affected side or fails to appear at all if the ureter is obstructed or the renal parenchyma is destroyed, as is the case in pyonephrosis.

Differential diagnosis. Acute renal colic is differentiated from acute appendicitis, cholecystitis, biliary colic, intestinal obstruction, perforating ulcer of the stomach or duodenum, and ptotic kidney. In cholecystitis, pains radiate to the shoulder-blade and there is tension of the abdominal wall in the region of the gallbladder. Stabbing pains, pronounced tension of the abdominal wall, and a history of peptic ulcer indicate the presence of perforating ulcer. In intestinal obstruction, dysuric symptoms are absent, the abdomen is inflated irregularly, and the urine is normal. A renal colic occurring in an upright posture and passing in a recumbent posture is characteristic of ptosis of the kidney.

X-ray diagnosis is of decisive importance in the period of remission of renal colic. A scout film of both kidneys, ureters and the urinary bladder makes it possible to determine the presence of a calculus or calculi, their form and size. The most intense shadow is produced by oxalate, phosphate, and carbonate calculi. Urate and cystine stones are not seen on an x-ray film. They are detectable only when they are of a mixed character. A contrast or retrograde intravenous roentgenography is employed for locating renal calculi. This method is used to determine the site of lodgement of stones in the pelvis, the calyx, and the ureter and to identify hydronephrosis, distention of the pelvis and ureter, congenital anomalies of the kidneys and ureters, etc.

Prophylaxis and treatment. For prophylactic purposes, it is necessary to eliminate all hindrances to a free drainage of urine (constriction, adenoma of the prostate, etc.) and to remove foci of infection from the teeth and tonsils. To prevent the formation of urate calculi, a diet of milk and vegetables and mineral water is prescribed. In the presence of oxalate calculi, restrictions are imposed on foods rich in calcium (milk, raw eggs, potatoes), with total abstinence from chocolate, spinach, gooseberries, and carrots. A patient with phosphorus-carbonate stones is kept on a meat diet and given hydrochloric acid and much water to drink.

The treatment of nephrolithiasis is aimed at the following goals: (1) to abort an attack of renal colic; (2) to facilitate the passage of smaller calculi with urine; (3) to remove larger calculi which fail to pass spontaneously; (4) to prevent the formation of new calculi.

An attack of renal colic may be aborted by the application of heat (hot-water bottle, or heater) to the lumbar region, a warm bath and administration of narcotics in combination with spasmolytic agents (1 ml of a 2 per cent solution of promedole, pantopon, a 1 per cent morphine solution with 1 ml of 0.1 per cent atropine sulphate or platyphylline). Lumbar blocking after Vishnevsky or novocain blocking of the spermatic cord in men and of the round ligament in women is also employed (20 ml of a 1 per cent or 40 ml of a 0.5 per cent novocain solution is injected into the depth of the spermatic cord; in women the same solution is injected into tissues adjoining the periosteum of the pubic bone and the labium majus pudendi at the site of attachment of the round ligament of the uterus). Should this prove ineffective, catheterization of the ureter is undertaken to displace the calculus and eliminate obstruction.

Stones of not more than 1 cm in diameter and rarely those of 2 cm in diameter pass spontaneously. To facilitate the passage of smaller stones vigorous walking, siphon enema, and subaqual irrigation are recommended, which intensify ureteral peristalsis and contribute to the passage of stones. In the presence of uric acid salts (urates, oxalates), the patient is enjoined to drink alkaline water in quantity and in the presence of phosphates, acid water.

For the same purpose, water purges are recommended; the patient is enjoined to drink 1.5 litres of water or tea twice weekly on an empty stomach in combination with spasmolytic drugs (atropine or platyphylline given three times daily per os or twice daily in injections). Stones may be removed with the aid of essential oils (terpenes), preparations of rovatin or rovatinex. A dose of 3-5 drops of rovatinex on sugar is prescribed to be taken 3-4 times during a period of several months. In case of calculi strangulation the single dose is increased to 40 drops.

In the majority of cases, ureteral calculi pass spontaneously; if, however, they fail to pass, catheterization of the ureter is carried out. In case of infected calculi, antibiotics (penicillin, levomycetin, synthomycin) are administered; for calculi of mixed origin, biomycin, terramycin, cycloserine, monomycin, and furadonin are helpful.

Indications and contraindications for surgery. Large isolated uninfected calculi should be removed by pyelotomy in order to avoid the possible risk of infection and death of the renal parenchyma. If a stone is small (1 cm), an operation may be unnecessary, because such stones pass spontaneously into the ureter and are expelled in the urine. A stone jammed within the ureter should be removed by any means (water purgation, spasmolytic agents, terpenes, catheterization, etc.), including surgical intervention if conservative therapy has proved unsuccessful. In case of pyonephrosis, giant hydronephrosis with atrophy of the renal parenchyma excision of the kidney and the ureters may be required. If, however, the stone is isolated, and the calyx is normal, one should abstain from an operation since the presence of such a stone does not adversely affect the patient's condition. In case of multiple infected stones in the calyces excision of the kidney is also undertaken.

Treatment at a health resort is prescribed with a view to promoting urinary secretion and the passage of mucus and leucocytes with urine.

TUBERCULOSIS OF THE KIDNEYS (TUBERCULOSIS RENUM)

Tubercle bacilli enter the kidneys from tuberculous foci (in the lungs, lymphatic glands), exclusively with the blood stream.

Pathologic anatomy. The kidneys affected by tuberculosis are enlarged. In the medullary substance there develop tubercles tending to cheesy disintegration and formation of cavities. In a disease running a progressive course, the affected kidneys become knobby. By this time the disease process extends to the pelves and later to the ureters and urinary bladder. In men, cheesy disintegration in the terminal stages is discovered in the seminal vesicles, testes and prostate.

Clinical picture. The disease may run its course without symptoms for a long period. By the time the renal pelves and urinary bladder are involved in the disease process signs of chronic pyelitis and cystitis may appear. Also at this time, nagging pains begin to be felt in the lumbar region or in the region of the urinary bladder. Painful (dysuria) and frequent (pollakiuria) micturition is noted. The temperature becomes subfebrile. The urine is turbid, with a substantial sediment and an acid reaction. Macroscopically, a large number of leucocytes and frequently erythrocytes are discoverable. Pus often found in acid urine is an important diagnostic sign of tuberculosis of the kidneys and urinary bladder, since in the presence of an ordinary purulent infection the urine reaction is alkaline.

Mycobacterium tuberculosis is very difficult to discover. For this purpose, inoculations of urine into special nutrient media are necessary.

Another method employed for identifying tuberculous infection is injection of the patient's urine into the abdominal cavity of a guinea pig. If tubercle bacilli are present in the urine, the animal will develop the disease within 6 weeks of injection.

An important role in the diagnosis of tuberculosis of the kidneys and urinary bladder is played by pyelography, chromocystoscopy, and catheterization of the ureters. When the pelves are filled with a contrast stain, jagged contours of the pelves and calyces and calcified foci resembling calculi are found in the x-ray film.

The disease usually lasts from 3 to 5 years. In unamenable cases it ends in death due to a tuberculous intoxication of the body, amyloid degeneration of the kidneys, uraemia or extension of the disease process to other organs.

Treatment. Today, an important remedy against tuberculosis of the urinary passages is streptomycin, especially when it is administered early in combination with general measures to strengthen the patient's health. Patients with a considerable tuberculous lesion in one kidney are subjected to surgical treatment, which consists in a total or partial resection of the kidney.

PYELITIS-PYELONEPHRITIS

Pyelitis, or pyelonephritis, may be acute or chronic, unilateral or bilateral, is sometimes accompanied by affection of the renal tissue, and when taking a chronic course leads to a secondary contraction of the kidney with azotaemia.

Acute Pyelonephritis (pyelonephritis acuta)

Aeliology and pathogenesis. Acute pyelitis (pyelonephritis) develops in influenza, enteric fever, furunculosis, chronic tonsillitis, osteomyelitis, septicopyaemia, etc. Pathogenic microorganisms enter the kidney or pelvis by a descending route, i.e., with the general blood stream. Acute pyelonephritis may also develop by an ascending route when pathogenic bacteria invade the pelvis and parenchyma of the kidney from the urinary bladder through the ureters. This is noted in cases of obstructed urinary drainage, congenital anomalies of the urinary passages, strictures, calculi, newgrowths in the ureters and rectum. An inflammatory process in the pelvis may also be provoked by penetration of microbes mainly *E. coli*, into the lymphatic system of the pelvis. This is favoured by the presence of a large number of anastomoses between the lymphatic routes of the ascending colon, the vermiform appendage and the ureters.

In acute pyelitis (pyelonephritis), the urine may show the presence of *E. coli*, enterococcus, *Proteus vulgaris*, *Staphylococcus aureus*, *Staphylococcus albus*, etc. The penetration of microorganisms into the renal pelves is not always accompanied by an inflammatory process. The requisite conditions for this are virulence of the bacterial flora and a certain susceptibility of the body itself. An important role in the development of pyelonephritis is played by a disturbance in the nervous regulation of urinary bladder function, which is found particularly often with various disorders of the central nervous system, lesions of the spinal cord and diabetes mellitus. Pyelonephritis is more frequent in women than in men. In men, the factors contributing to the development of pyelonephritis may be diseases of the prostate and nephrolithiasis. In women, pyelonephritis is particularly often found in pregnancy, possibly in connection with retention of the urine due to compression of the ureter by the enlarged uterus. The right kidney is more liable to affection than the left. This is accounted for by the fact that normally the right kidney is situated lower than the left, which contributes to urine retention within it. A certain role is played by the common lymphatic system of the right kidney, the ureter and the ascending colon.

Pathologic anatomy. The kidney is somewhat enlarged, the pelvis is distended, the mucous membrane of the pelvis is hyperaemic, oedematous, loosened, at places ulcerated and covered with purulent secretions. Microscopy reveals invasion of the pelvic wall by leucocytes, pus in the tubules, and infiltration of the intertubular connective tissue with leucocytes. In the cortex and medullary substance of the kidneys multiple abscesses are found sometimes. Bacteria are discovered in the tubules and the interstitial tissue of one or both kidneys.

Clinical picture. Acute pyelitis sets in with a chill and a temperature rise to 40°C and higher. The fever is intermittent or continuous. The patient complains of headaches, palpitation, dyspnoea, nausea, vomiting, and frequent micturition. Acute and sometimes dull lumbar pains appear, which are due to distention of the renal pelves with exudation. They grow worse on walking, movement and light strokes applied to the kidney region (Pasternatsky's sign).

Acute pyelitis is accompanied by neutrophilic leucocytosis with prevalence of rod neutrophils and an elevated ESR. The most characteristic sign is pyuria, microhaematuria and macrohaematuria, particularly with a coexisting cystitis. The protein level in the urine does not exceed 1 pro mille. As a rule, in pyelitis there is no oedema, hypertension, and the urinary sediment contains no casts. Roentgenography reveals deformity and distension of the pelves and calyces.

In pyelitis, tubular function is the first to suffer because oedema and cellular infiltration developing between the tubules cause compression of the tubular epithelium and damage to the blood vessels which supply them. Water reabsorption and the specific gravity of urine are reduced. At the same time, in pyelitis running a prolonged course there is a reduction in the filtration capacity of the glomeruli, which is responsible for the increase in nonprotein nitrogen in the blood and the development of uraemia.

Acute Pyelonephritis in Pregnancy

Acute pyelonephritis in pregnancy may sometimes run its course without symptoms of dysuria, fever, lumbar pains and marked pyuria. In such cases, a timely identification of the disease is facilitated by urinalysis with estimation of the white cell count in the urine sediment and by bacteriologic examination. Unidentified pyelitis in pregnancy may lead to chronic pyelonephritis.

Acute pyelonephritis treated in due time ends in complete recovery. In one out of every four cases, the disease may take a chronic course.

Prophylaxis and treatment. Prophylaxis consists in measures to control infection and prevent obstructions of urinary drainage. Highly important are healing procedures in the oral cavity, the fauces, and the paranasal sinuses, a vigorous treatment of inflammations in the urinary passages and the genital system, as well as of polyps and constipation. Maximum caution should be displayed in catheterization of the ureters and in other urologic examinations lest infective matter be introduced by accident.

Treatment. Agents effective against infection and inflammation (penicillin, streptomycin, terramycin, tetracycline, etc.) and chemical drugs (norsulphazol, sulphadimezin, urosulphane in a dose of 3-4 g daily during a period of 10-15 days) are administered. The treatment may be discontinued only after 2-3 urinalyses have revealed normal values. Levomycetin (synthomycin) is also prescribed in a dose of 3-4 g to be taken daily during a period of 7-10 days. During therapy the patient is confined to bed and is kept on a milk and vegetable diet. Spicy and pungent foods (pickles, marinades, smoked foods) are forbidden. It is recommended to drink liquid in quantity (tea with lemon, mineral water). Applications of heat to the lumbar region are also helpful.

Chronic Pyelonephritis (pyelonephritis chronica)

Chronic pyelonephritis is a sequel to untreated acute pyelonephritis, diseases disturbing urinary drainage (strictures of ureters, nephrolithiasis, hydronephrosis) and inflammations in the urinary bladder and the urethra (cystitis, prostatitis, urethritis, etc.), as well as neurologic diseases (of the spinal cord, etc.). The organisms which are most commonly cultivated from urine are *E. coli* and enterococci as well as staphylococci, streptococci, *Proteus vulgaris*, and sometimes gonococci.

Pathologic anatomy. The pathologic process extends from the pelvis and medullary substance to the renal cortex. Autopsy reveals that the kidney is contracted, knobby and considerably diminished in size. If contraction is absent, there is an inflammatory infiltration of the interstitial tissue, mainly in the intertubular spaces. In the period of kidney contraction there is interstitial fibrosis. Chronic pyelonephritis is also characterized by atrophy of the tubules, the appearance of colloid casts in the urine, and in certain cases necrosis of the pelves, infiltration and connective tissue growth in the renal capsule.

Clinical picture. Chronic pyelonephritis occurs in several forms: latent, cystopyelitic, and contractive. The most frequent is the latent form, which is subsequent to acute pyelitis or runs an insidious course from its very onset. In the absence of an exacerbation this form produces no symptoms typical of pyelitis, i.e., fever, leucocytosis, an elevated ESR, and hypertension. Anaemia, headaches, symptoms of dysuria, and oedema are also absent. There is only proteinuria within the limits of 1 pro mille, slight haematuria and cylindruria. The most characteristic sign of the latent form of pyelonephritis is pyuria (from women urine should be collected with a catheter). Sometimes pyuria may be absent if the disease process is localized in the interstitial tissue.

The cystopyelitic form of chronic pyelonephritis runs its course like acute pyelitis, with periodic exacerbations, symptoms of dysuria, lumbar pains, fever, leucocytosis and, what is the most important, with pyuria and proteinuria. Chronic pyelonephritis usually ends in renal insufficiency, hypertension and azotaemia. In such cases, the clinical course of pyelonephritis resembles that of kidney contraction in hypertensive vascular disease and chronic nephritis.

Renal insufficiency in chronic pyelonephritis is distinguished by a number of specific features. A characteristic sign of this condition is an irregular affection of the kidneys. It is often localized only in one kidney. Since the inflammatory process arises in the intertubular tissue with involvement of the vessels which supply the tubules, an impairment of function is at first seen in the tubular apparatus and later in the glomeruli. In this connection, reabsorption is initially reduced in the tubules and polyuria develops in combination with hyposthenuria and pyuria. The concentrating power of the kidneys is disturbed very early, but renal insufficiency makes its appearance slowly.

Hypertension is more frequently encountered in bilateral pyelonephritis but it may also be present in a unilateral affection of the kidneys. In the majority of cases, hypertension runs a benign course, less often a malignant one, and tends to progress very slowly. The development of hypertension is accompanied by characteristic symptoms: headache, changes in the fundus oculi, hypertrophy of the left cardiac ventricle, etc. Hypertensive crises, however, and lesions of the coronary and cerebral vessels are observed very rarely. Upon recovery from chronic pyelonephritis arterial hypertension returns to normal. Oedema is encountered rarely, and is mostly associated with myocardial insufficiency.

Prognosis has become more favourable during the past few years owing to a highly effective treatment (excision of a kidney, antibiotic and chemotherapy, etc.) which prevents the development of renal insufficiency.

The *diagnosis* of chronic pyelonephritis rests on the above-mentioned clinical symptoms as well as on a special examination of the urinary sediment by employing the Kakovsky-Addis method, urography, and biopsy of the kidneys. According to the Kakovsky-Addis method, the urine is collected during 10 hours, stirred, measured for volume, after which one-fifth of an hour's portion is taken for centrifugation. In the test tube, 0.5 ml of urine with sediment is allowed to remain. In the layer having no sediment the quantity of protein is determined, and the total amount of protein in the urine discharged per 24 hours is calculated in grams. The sediment is shaken and transferred with a pipette into a chamber in which leucocytes, erythrocytes and casts are counted. The leucocytes and erythrocytes are counted in 15 large squares and the resulting sum is multiplied by 1,000,000. The tube casts are counted in 15 squares, the sum being multiplied by 100,000. In this way the number of blood corpuscles in the urine secreted per 24 hours is determined. Normally, the number of leucocytes in urine secreted in 24 hours is 2,000,000, erythrocytes, 1,000,000, and casts 2,000. Chronic pyelonephritis is characterized by a considerable increase in the white cell count, whereas chronic nephritis produces an increase in the red cell count and to a lesser extent in the white cell count.

Urography. For diagnosis of chronic pyelonephritis, intravenous and retrograde pyelography is used, sometimes simultaneously with pneumorenography. Chronic pyelonephritis causes a characteristic deformation of the renal cavities manifested by distention of the pelves, calyces and narrowing of their isthmus. In the early period, local spasms of the calyx or pelvic system may be discovered. Contraction of the kidneys causes an irregular diminution of their size and ruggedness of their contours.

For identifying unilateral chronic pyelonephritis, it is recommended to use, in addition to x-ray photography, catheterization of the ureters and to examine the content of protein and blood corpuscles in the urinary sediment.

Biopsy. In diagnosis of chronic pyelonephritis, biopsy of the kidneys is used at present.

Prophylaxis and treatment. For prophylactic purposes, stones and tumours are removed and constrictions eliminated by an incision. Highly important is also treatment of inflammatory processes in the urinary tract, urethritis, cystitis, acute pyelitis, pyelonephritis, and various infectious foci (tonsillitis, appendicitis).

The treatment of chronic pyelonephritis consists above all in eradication of the causes of inflammation. For this purpose, it is sometimes enough to eliminate obstacles to the urinary drainage by liquidation of strictures, removal of stones and sometimes of a kidney, in particular in case of the latter's contraction if the affection is unilateral. In the period of exacerbation, chemical drugs and antibiotics (sulphathiazole, sulphazole, urosulphane at first in doses of 5-6 g daily, and then in daily doses of 2-4 g during 3 weeks, levomycetin in a dose of 0.5 g four times daily, penicillin, streptomycin, biomycin, tetracycline, etc., in doses of 400,000-800,000 U daily) are used. Chemo- and antibiotic therapy should be continued with a check-up on the condition of the urine until pyuria disappears and the urine becomes sterile. Chemical drugs and antibiotics do not always eliminate pyelonephritis but at least produce an abatement of the inflammatory process. Treatment with an artificial kidney has failed to give a lasting effect. In the treatment of hypertensive syndrome the greatest importance is attached to the liquidation of pyelonephritis. A considerable amelioration in the patient's condition may take place under the influence of hypotensive remedies (magnesium sulphate, serpasil, raunatin, reserpine, dibazol, etc.).

INFLAMMATION OF THE URINARY BLADDER (CYSTITIS)

Inflammation of the mucous membrane of the urinary bladder results from the entry of infection with the blood stream (in typhus, influenza), lymph flow (gonococcus, staphylococcus, streptococcus, *E. coli*) or through the ureters in tuberculosis of the kidneys, pyelitis (tubercle bacilli, *E. coli*, staphylococcus, streptococcus). The urinary bladder may become infected in case of violation of the rules of antisepsis and asepsis during its catheterization. Inflammation of the urinary bladder also develops in compression of the urethra resulting from hypertrophy of the prostate in patients ridden to bed for a long time, or in paralysis of the bladder, with prolonged retention of urine and infection within it.

Clinical picture. Cystitis is known to occur in an acute and chronic form. Acute cystitis at the onset of the disease is accompanied by a rise in temperature and frequent, painful micturition (dysuria). Dull and lancinating pains are felt in the lower part of the abdomen, which radiate to the urethra. In frequent micturition only several drops of urine are discharged at a time, which cause burning pain in the urethra. The urine is turbid, contains many leucocytes, epithelial cells, erythrocytes, gives an alkaline reaction and has an ammonia odour. An acute attack runs a moderately long course, the disease lasting 2-3 weeks. As has been pointed out above, the infection may spread from the urinary bladder through the ureters into the renal pelves (ascending infection). In chronic cystitis the cardinal symptom is the presence of a large number of leucocytes in the urine; other symptoms are indistinct or absent.

Treatment and prophylaxis. It should be kept in mind that among the frequent causes of cystitis are various prolonged and severe diseases, sometimes inexpertly performed catheterization, and the patient's own uncleanliness. Therefore, a correct management of the patient is sometimes by itself enough for the prevention of cystitis. In acute retention of the urine in the event of any infectious disease in the postoperative period it is necessary to restore spontaneous micturition, and only in case of failure resort to catheterization. At first a hot-water bottle is applied to the lower abdomen, which usually causes passage of urine.

Catheterization is carried out with strict observance of the following rules: the catheter, preferably elastic, must be absolutely sterile and placed before catheterization on a sterile trough in which sterile cotton tampons soaked in corrosive sublimate solution and a sterile dissecting forceps are also kept. One intending to perform catheterization should have his hands carefully washed with warm water and soap and wiped with alcohol, a 5 per cent lysol solution or a 2 per cent chloramine solution. Then the glans penis (in men) or the region of the outer orifice of the urethra (in women) is twice wiped with tampons soaked in corrosive sublimate solution. Before insertion into the urethra, the end of the catheter is greased with sterile petrolatum oil and slowly, without effort and pressure, slipped into the urinary bladder. The passage of the catheter through the sphincter of the bladder is felt by the patient.

The treatment of acute cystitis is carried out in conditions of bed rest. The patient's diet should contain no foods likely to irritate the urinary passages. The patient is given per os salol (phenyl salicylate) with belladonna and a tincture of bear berry (formula No. 50) which promotes the eradication of infection by its strong diuretic effect. Urotropin (methenamine), streptocid (sulphanilamide), and penicillin are also prescribed. Patients with long-standing cystitis have the urinary bladder irrigated with a 1 : 1,000 rivanol solution or a 0.25-0.5 per cent silver nitrate solution.

Diseases of the Digestive Tract

DISEASES OF THE OESOPHAGUS

Anatomic and physiologic outline. The oesophagus is a continuation of the pharynx. It begins at the level of the cricoid cartilage opposite the body of the 6th cervical vertebra, descends along the anterior surface of the spinal column, and opens out into the stomach at the level of the 11th or 12th thoracic vertebra. The oesophageal wall comprises three coats: the mucous membrane (inner coat), the muscular layer (middle coat), and the serous membrane (outer coat); it is enveloped by a layer of loose connective tissue. There are three physiologic narrowings: (1) at the very entrance to the oesophagus; (2) behind the bifurcation of the trachea into two bronchi at the level of the 4th thoracic vertebra, and (3) at the site of the oesophagus piercing the diaphragm at the level of the 10th thoracic vertebra. The oesophageal wall is not more than 2-3 mm thick. The mucous membrane consists of longitudinal folds harbouring mucous glands. The muscular layer consists of internal circular and external longitudinal fibres. The oesophagus is supplied with blood by the inferior thyroid, subclavian, oesophageal, bronchial, left gastric, and inferior phrenic arteries. It is innervated by the sympathetic and parasympathetic nerves.

The oesophagus conveys liquid food from the oral cavity into the stomach without the aid of the musculature; solid food, by means of an active peristalsis of the muscular layer.

Methods for examining the oesophagus. Examination of the patient is begun with consideration of complaints, collection of evidence on the case and life history, and is rounded off by an objective study. Patients with diseases of the oesophagus complain of dysphagia (difficult deglutition), pains felt along the course of the oesophagus, and "oesophageal" vomiting. Questioning the patient helps locate the site of dysphagia and pains, the routes of their radiation, a sudden or gradual appearance of abnormal sensations, association of the latter with the character and consistency of food and with physical or nervous strain. The nature of vomited matter has also to be clarified, since oesophageal vomit, as distinct from gastric vomit, contains a large quantity of undigested food. It is free of hydrochloric acid as well as pepsin.

Objective findings. The object of examination is not only the oesophagus but also the adjacent organs: the lungs, bronchi, heart, aorta, mediastinum and stomach. Attention is given to deglutition murmurs and to the condition of the neck and the upper half of the thorax, where a dilatation of veins due to a circulatory disturbance in the mediastinum may be observed. The examiner should determine the sensitivity of the skin of the neck by palpation, the presence of oedema, infiltrations, fluctuation or pasty swellings. An objective study of the oesophagus is completed by oesophagoscopy and probing of the oesophagus.

Oesophagoscopy. The oesophagoscope consists of a hard metal outer tube, 25 cm in length (for adults) and 14 mm in diameter, an inner tube of the same length and a lighting system. For oesophagoscopy the patient is put in a recumbent position, usually on his right side, the right leg being flexed at the knee and the left leg extended. Before introducing the oesophagoscope, the mucous membrane of the root of the tongue, the epiglottis and the oesophageal orifice is painted with a 10 per cent solution of dicaine with adrenalin or a 3 per cent dicaine solution. The oesophagus is examined twice: during insertion of the tube and during its slow withdrawal. Oesophagoscopy should be undertaken in an oesophageal disease of obscure aetiology, obstruction of a sudden onset, and when it is necessary to remove a foreign body. The principal contraindications for the procedure are a burn of the oesophagus in the first 7-10 days of the accident, tuberculosis of the larynx and lungs with destruction of tissue, aortic aneurysm, and a circulatory disturbance. In oesophagoscopy, air is pumped into the oesophagus to produce its distention. This method is usually employed in biopsy or extraction of foreign bodies. Oesophagoscopy can also be performed through a gastroscopic opening. This is done in cases of organic or spastic strictures impassable from above, for foreign bodies of low localization, and multiple cicatricial changes in the oesophagus.

Probing of the oesophagus. In view of the great likelihood of perforation of the oesophagus, probing is very rarely undertaken at present. Usually bouginage of the oesophagus is performed for the sake of its dilatation and prevention of strictures likely to form at the site of burns caused by concentrated alkaline or acid solutions. Probing should be preceded by x-ray examination of the oesophagus and oesophagoscopy is also advisable. The bougies are 70 cm long and vary in thickness, beginning from one as thin as a thread. Before use, a bougie is immersed in warm water and greased with some oil. The constricted portion is dilated gradually and slowly by consecutive insertion of several bougies of a successively increased diameter. Bouginage is performed once in every one or two days during a period of 1-3 months.

Developmental Anomalies of the Oesophagus

Congenital anomalies are sometimes encountered, such as underdevelopment, stricture, distention and elongation of the oesophagus, and an oesophago-tracheal fistula. In case of its underdevelopment (atresia) the oesophagus turns into an impassable fibromuscular tube, whereas in a subject with a short oesophagus supradiaphragmatic hernia develops. Developmental anomalies of the oesophagus present a corresponding clinical picture, but a role of decisive importance, however, is played by x-ray examination and oesophagoscopy. Congenital stricture of the oesophagus readily responds to bouginage, other cases of oesophageal maldevelopment and oesophago-tracheal fistula require surgical intervention.

Diverticulum of the Oesophagus

Diverticulum of the oesophagus is a circumscribed bulging of the oesophageal wall, sometimes involving all its layers. Pulsion, traction and pulsion-traction diverticula are distinguished. Pulsion diverticula are caused by bulging of the oesophageal wall from the inside and, with regard to their location, are subdivided as follows: (1) high, pharyngo-oesophageal, ranging in size from a pea to a child's head; (2) oesophageal.

Clinical picture. The initial complaints are those of a smarting sensation in the throat, eructation after meals, and vomiting. These are later joined by difficulty in passage of now the first, now the last mouthfuls of solid and liquid food. With a diverticulum of a considerable size, the neck appears to be enlarged. On percussion of this region a dull sound is elicited, and on palpation, air with food pulp is expressed. The patient often complains of dyspnoea caused by pressure of the diverticulum on the trachea and the vessels of the neck. Venous stasis is observed simultaneously. In x-ray examination, the contrast substance, in case of evacuation of the food mass from the diverticulum, accumulates at the base of the neck or somewhat higher. After the diverticulum has been filled, barium descends lower; therefore beside a shadow from the full diverticulum a band of barium descending into the oesophagus is visible. The disease tends to progress and the patient is losing flesh. In compression by a diverticulum of the organs located in the region of the neck, lymph stasis, venous stasis, neuralgia and attacks of angina pectoris may ensue.

Epibronchial (at the level of the tracheal bifurcation), epinephral (above the diaphragm), epicardial (above the cardia), and pharyngo-oesophageal diverticula are commonly found in men of 40-60 years of age and are localized in the anterior and anterolateral wall. Complaints of retrosternal pains, oesophageal vomiting, palpitation, attacks of angina pectoris and difficulty in breathing appear only when the diverticulum is considerably enlarged and the passage of food is difficult. The diagnosis is verified by means of x-rays and oesophagoscopy (Fig. 50).

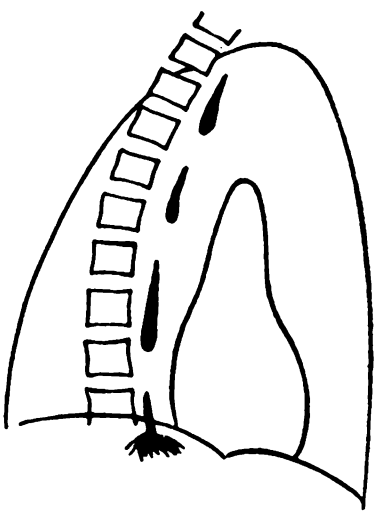

Fig. 50. X-ray of the oesophagus

Traction diverticula (Lat. *trahere* — to draw) are more frequent than pulsion diverticula. They develop in adhesions of the outer membrane of the oesophagus to inflamed lymphatic glands of the mediastinum, in mediastinal pleurisy, bullet wounds of the thorax, etc. In such cases the oesophageal wall is drawn to the affected side to form a diverticulum shaped like a pouch or funnel. Traction diverticula cause little discomfort to the patient because food is rarely retained within them, although cases are known of perforation and abscess formation.

Treatment. In the presence of pulsion and traction diverticula, coarse food and alcohol are forbidden. The patient is recommended to wash down food with warm alkaline (mineral) water. The surgical treatment of diverticula consists in their excision.

Achalasia (Cardiospasm) and Idiopathic Distention of the Oesophagus

Achalasia results from a disturbance during a meal of the rhythmic contraction of the oesophagus, which is regulated by the vegetative nervous system—the sympathetic and vagus nerves. The development of achalasia is due to general and local factors. Among the former are nervous shock, prolonged emotional upset; among the latter, mechanical, chemical, and thermal irritation of the oesophagus and stomach. A contributing factor of smaller importance is malnutrition (vitamin deficiency and dystrophy of the oesophageal nerve plexuses), as well as diseases of the adjacent organs—the stomach, gallbladder, etc., which cause cardiospasm of reflex origin.

Clinical picture. The disease affects the ages of 20 to 40 years. The patient complains of food retention behind the sternum and in the lower part of the oesophagus after ingestion of solid, less often liquid, food, as well as cold water and aerated drinks. The patient is afraid to eat. Later, these symptoms are joined by regurgitation of food. The difficulty in the passage of food is at first periodical, and then becomes permanent. To facilitate the passage of food the patient has to drink water after each swallow. Stagnation of food in the oesophagus may cause vomiting of undigested material, mucus, the vomit is sometimes foetid, and contains no hydrochloric acid or pepsin. There may also be an inflammation of the oesophagus, with erosions, ulceration and scar tissue formation. The oesophagus is diffusely dilated, its holding capacity is increased to 2-3 litres (compared to the normal 200 ml) and its diameter grows to 12-13 cm. Pressure of the dilated oesophagus on the adjacent organs gives rise to cyanosis, dyspnoea, attacks of angina pectoris, etc. The patient becomes emaciated, develops bronchitis, bronchiectasis in consequence of aspiration of gastric contents, and constipation. On auscultation of deglutition the second deglutition murmur produced by food entering the stomach is delayed or absent. The left side of the neck is deformed and swollen. X-rays show a widening and elongation of the mediastinal shadow, the contrast substance is long retained in the dilated portion of the oesophagus, and the folds of the mucous membrane relief are thickened. In oesophagoscopy, the oesophagoscope tube rebounds within the dilated part of the oesophagus.

Course. Achalasia runs its course with periods of aggravation and remission. It is known to occur in two forms—compensated and decompensated, the former meaning achalasia without dilatation of the oesophagus, and the latter, with its diffuse dilatation. Achalasia must be differentiated from oesophageal cancer, whose characteristic signs are rapid progression of the disease, aggravation of dysphagia, an elderly age of the sufferer, absence of any considerable dilatation of the oesophagus, and typical x-ray findings. If treatment is not belated, the prognosis is favourable.

Treatment. In achalasia, treatment consists of mental rest and a rational diet. Food must be bland with respect to its mechanical, chemical and thermal effects. It should be thoroughly chewed and taken in small portions. Spasm is eliminated by administrations of atropine, platyphylline, papaverine, as well as by lavage of the oesophagus with warm water, a camomile infusion, a 2 per cent borax solution, or a 0.02 per cent silver nitrate solution. Vagosympathetic blocking, vitamins B_6 and B_1 are also prescribed. In case of severe emaciation, an anastomosis is established between the fundus of the stomach and the dilated part of the oesophagus. Plastic surgery of the cardia is also recommended. The disease runs a prolonged course, and patients live to a very old age.

Inflammation of the Oesophageal Mucosa (oesophagitis)

Oesophagitis develops in infectious diseases (measles, influenza, scarlet fever, etc.), after a slight injury, scratches, ingestion of very hot food and liquids, and commonly as a result of burns due to acids and alkali. Sometimes oesophagitis is secondary to the entry of infection from the oral cavity, the pharynx (dental caries, tonsillitis) into the oesophagus.

The gravity and extension of oesophagitis resulting from a burn of the oesophagus caused by concentrated solutions of caustic alkali and acids depend on the nature of the chemical agent (the most severe lesions are caused by sulphuric and nitric acids), its concentration, and duration of action on the oesophagus.

Pathologic anatomy. The characteristic signs of oesophagitis are hyperaemia, looseness and oedema of the mucous membrane, and the formation of superficial and deep ulcers, granulations and scar tissue. The inflammatory process may extend to the adjacent organs, and the oesophagus may become perforated. The inflammation is confined mainly to the sites of physiologic narrowings of the oesophagus. In case of a deep affection of the oesophagus abscesses form both in the oesophagus itself and in the surrounding cellular tissue. The formation of multiple scars causes complete obstruction of the oesophagus.

The *clinical picture* of oesophagitis varies with the depth of extension of the inflammatory process in the oesophagus. The mildest course is run by the superficial form. Its symptoms are a burning pain, pains about the neck and behind the sternum during food passage through the oesophagus, salivation, and reflux of the stomach contents. The disease is not lasting and its outlook is favourable. In corrosive oesophagitis the patient complains of excruciating pains along the course of the oesophagus, in the chest, and in the epigastrium, which radiate to the back, of salivation, painful eructation, vomiting of sanguineous material, and thirst. Deglutition is impossible. The oral mucosa and tongue are hyperaemic, with pallor of the skin, and cold perspiration on the face. Tachycardia, fever, hypotension, small pulse, and neutrophilic leucocytosis appear. The urine contains protein, erythrocytes, leucocytes and casts. Corrosive oesophagitis may be complicated by oedema of the epiglottis, larynx, and trachea, and also by pneumonia, if caustic fluid enters the respiratory tract, as well as by perioesophagitis, peritonitis, perforation of the oesophagus, mediastinitis, pleurisy, and pericarditis. Death occurs within the first 24 hours from shock or a burn of the bronchi, and at a later time from perforation of the oesophagus, peritonitis, or pneumonia.

The diagnosis is based on evidence obtained from the patient and his relatives, examination of the remains of the material responsible for the accident, the odour emitted from the patient's mouth, as well as an examination of the mucous membranes of the lips, oral cavity and pharynx. Poisoning with acetic acid leaves whitish-grey patches; with nitric acid, yellow and greenish-yellow scabs; with carbolic acid, snow-white scabs; with sulphuric and hydrochloric acids, greyish-white scabs.

If the disease takes a favourable course, pains gradually subside, and the patient feels better on the 8th-10th day after the accident. By the same time, the temperature returns to normal and the working capacity is quickly regained. In case of marked oesophagitis and incorrect therapy stenosis of the oesophagus and stomach ensue.

Prognosis depends on the concentration and quantity of a poison and the duration of exposure of the oesophagus to it.

Treatment and prophylaxis. For superficial oesophagitis a diet of liquid lukewarm food is prescribed: butter, milk cream, thin gruels, whipped white of egg, soft-boiled eggs, almond milk, and sieved vegetables. The drug therapy consists of administration of a 0.5 per cent novocain solution per os in a dose of one tablespoonful 3-5 times daily, anaesthesin in a dose of 0.5 g 2-3 times daily, or, preferably, a mixture consisting of 3 g of anaesthesin and 200 ml of olive oil in a dose of one tablespoonful 3-4 times daily.

For prophylaxis of superficial oesophagitis, one is recommended to eat unhurriedly, thoroughly chew the food and abstain from very hot or very cold food and drink.

The treatment of corrosive oesophagitis is more complicated. In the first place urgent measures are taken against shock and to remove poison from the patient's system. Pains are alleviated by subcutaneous injections of atropine, morphine, while poison is urgently eliminated or diluted by washing out the oesophagus and stomach with 8-10 litres of lukewarm water introduced in small portions. To forestall a secondary infection, antibiotics (penicillin, streptomycin) are administered. During the first 24 hours, at least 3 litres of liquid, including a 5 per cent glucose solution, are introduced subcutaneously, intravenously and through an enema. ACTH is prescribed in a dose of 20 U to be given three times a day, as well as prednisone, prednisolone (in doses of 20 mg 3 times daily) during a period of 5-7 days. The remedies given per os are peach or olive oil with anaesthesin (3 g of anaesthesin and 200 g of olive oil) in a dose of one tablespoonful to be taken once in every 3 hours, almond emulsion with amygdalin (Emulsio Amygdalarum dulcium—200.0, Amygdalinum—0.1) in one tablespoonful dose 5-6 times daily, and a 0.5 per cent novocain solution in one tablespoonful dose every two hours. To reduce intoxication, 30-40 ml of a 40 per cent glucose solution with 5 ml of a 5 per cent ascorbic acid solution is administered intravenously. Camphor, cordiamine, and mesaton are prescribed for cardiovascular insufficiency. In the first two days of the disease the patient is given cool liquid food: ice cream, milk cream, milk, and later mucilaginous decoctions (diet No. 1*a*). Bouginage is begun on the 7th-10th day of the disease, at first with a soft and then with a hard bougie which upon insertion is left in place for 15-30 minutes. Bouginage is repeated every other day. Bougies of diameters consecutively increased to 10-12 mm should be employed. In case of phlegmonous oesophagitis, antibiotics are prescribed in large doses. Should medicinal therapy fail, surgery is indicated.

Benign tumours. Among benign tumours are warts, papillomas, lipomas, adenomas, fibromas, haemangiomas, lymphangiomas, cysts, etc. Fibromas, polyps and cysts are the most common.

The clinical picture varies with the size of a tumour. Large tumours cause dysphagia, vomiting, and impairment of nutrition. X-rays reveal a filling defect with even and distinct contours and a barium "streamline" symptom. The diagnosis is verified by means of oesophagoscopy and biopsy. Small pedunculated tumours are removed through an oesophagoscope with a galvanocaustic snare; large tumours require external oesophagotomy.

Malignant tumours. Oesophageal carcinoma is the most common malignant tumour of the oesophagus. Its incidence is higher in men, mainly at ages of 50 to 65 years.

The *aetiology* of oesophageal carcinoma is unknown. The factors contributing to this disease are various irritations: very hot food and drink, scars left by burns of the oesophagus, peptic ulcer, syphilis and tuberculosis of the oesophagus. The newgrowth is commonly localized at the level of the thyroid cartilage, the tracheal bifurcation and at the cardia, i.e., at the levels of the physiologic narrowings of the oesophagus.

Pathologic anatomy. Oesophageal carcinoma develops from the stratified squamous epithelium lining the mucous membrane. The disease is known to occur in three forms: (a) scirrhous; (b) cerebroid-ulcerative; and (c) villosum. Scirrhus is a squamous cell carcinoma and extends both longitudinally and circularly. The newgrowth encroaches on the surrounding tissue, and metastases are late in development. The cerebroid-ulcerative and the villosum forms of carcinoma are rarely encountered, resemble a cauliflower in outward appearance, and are liable to ulceration and decomposition.

Oesophageal carcinoma is characterized by the development of metastases to lymph nodes as well as by a direct extension to the adjoining tissues and organs. This is followed frequently by perforation, suppuration, infection, formation of an oesophago-tracheal or oesophago-bronchial fistula leading to aspiration pneumonia, suppurative processes in the lungs (abscesses and pulmonary gangrene), and putrid purulent mediastinitis. There have been cases of destruction of a large vessel resulting in a lethal haemorrhage, and paralysis of the lower extremities due to compression of the vertebral bodies or the spinal cord (Fig. 51).

Clinical picture. The patient at first complains of difficulty in swallowing, felt periodically, oesophagospasm which is due to eating solid food and cancer cell infiltration, and later, of a constant sensation of a foreign body present in the oesophagus. As the disease progresses the patient feels difficulty in swallowing not only solid but also finely pureéd food as a result of oesophageal stricture.

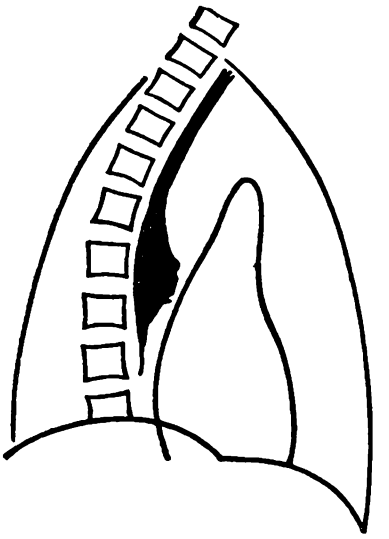

Fig. 51. X-ray of oesophageal carcinoma

These symptoms are joined by pains caused by the turmour encroaching on the surrounding organs and tissues. Dysphagia entails vomiting, sometimes of sanguineous material, while putrefactive processes occurring in the oesophagus cause a foetid odour from the mouth. In paresis of the left vocal cord due to extension of the newgrowth to the recurrent nerve the voice becomes coarse. Hard supraclavicular nodes become palpable rather early. As the disease progresses the patient grows thin because of malnutrition and water deficit. He suffers from thirst, is pale, and has oliguria with an increased specific gravity of urine. The blood shows hypochromic anaemia (at later periods, erythrocytosis and polyglobulia due to an increase in blood density), a diminution in the number of eosinophils and an elevated ESR. X-rays show an accumulation of contrast substance before the narrowed part of the oesophagus. The contrast substance at this site forms a narrow band. In the region of the stricture, irregularity of the mucosal relief, ragged or flattened contours of the folds of the oesophageal mucosa are determinable. In the presence of a fistula, barium masses may enter into the air passages. Death occurs within a period ranging from 3 months to 2 years and longer (in case of scirrhus). A frequent cause of death is bronchopneumonia.

Treatment. A radical method of treatment is resection of the affected part. Therapy includes general measures to improve the regimen, dietotherapy, drug therapy, radiotherapy, and dilatation of the oesophagus. Depending on the patient's condition, he is enjoined to have complete or partial bed rest. The diet must be nutritious and contain at least 100-120 g of protein, fats, and carbohydrates, as well as salts and vitamins. Food must be eaten in small portions and contain no chemical or mechanical irritants. In case the passage of food is difficult, the patient is maintained on a liquid diet. At least one litre of liquid must be drunk daily. If liquid passes through the oesophagus with difficulty, it may be injected subcutaneously as well as in microenemas of a 10 per cent glucose solution in doses of 200 ml twice daily. In case of a growing obstruction bouginage is undertaken at a medical institution and under the guidance of roentgenoscopy or oesophagoscopy in view of the possible risks of perforation of the oesophageal wall. Another remedy is gastrostomy, which serves to improve the patient's nutrition and subjective feeling. The rubber tube is periodically withdrawn from the stomach, washed, greased with petrolatum oil and reinserted into the opening. It should be remembered that this procedure should not last longer than 10-15 minutes, because otherwise the opening will soon begin to close and will have to be dilated, which is painful. The skin round the fistular opening is often macerated with gastric secretion; therefore, it should be painted with petrolatum oil. Sedatives are also prescribed (bromide, aqueous infusion of valerian, barbiturates), as well as antispastics (atropine, platyphylline), analgetics (anaesthesin, novocain, etc.), and vagosympathetic blocking. Radiotherapy fails to produce complete recovery and gives in some cases nothing more than a remission.

DISEASES OF THE STOMACH

An Outline of Anatomy and Physiology

The stomach, in its greater part, lies to the left of the median line and only its pylorus is in the right half of the abdominal cavity. The stomach is shaped as a hook or a curved pear. Its holding capacity is normally equal to 1,000-1,500 ml, but may be larger or smaller.

The stomach is a hollow muscular organ composed of the cardiac part, the fundus, the body and the pyloric part. The cardiac part adjoins the site of communication of the oesophagus with the stomach. The fundus (fornix) is the part under the cupola of the diaphragm to the left of and above the cardia. The body (or corpus) is the part below the fundus. The pyloric part is to the right of the median line and ends in the pylorus separating the stomach from the duodenum. Between the cardia and the pylorus lie the upper concave surface known as the lesser curvature (facing to the right towards the liver), and the lower convex surface known as the larger curvature. The stomach has an anterior and posterior wall and is kept in place by the oesophagus and ligaments consisting of double folds of the peritoneum.

The stomach wall has three coats: serous, muscular, and mucous. The serous coat covers the stomach on all sides on the outside. The muscular coat consists of three layers (outer, middle, and inner). The outer layer is made up of longitudinal, the middle layer, of circular, and the inner layer, of oblique muscle fibres. This enables the stomach to push food into the pyloric part (longitudinal fibres), ground (circular fibres) and mix it (oblique fibres).

The mucous coat consists of longitudinal and transverse folds. The former pass mainly along the lesser curvature forming a furrow known as the gastric canal, through which liquid food enters the pyloric part of the stomach. Denser food particles settle in the fundic part of the stomach.

The mucous coat lodges glands secreting the enzyme pepsin, hydrochloric acid, and mucus. The glands of the fundus have the principal (or adelomorphous) cells secreting pepsin and the parietal (or delomorphous) cells secreting hydrochloric acid. The glands of the pyloric part contain only the principal cells.

The stomach arteries take their origin from the celiac artery whose branches supply the stomach. The latter also receives branches from the splenic artery. From the stomach venous blood enters the system of the portal vein. Therefore, stasis in the portal vein causes a varicose dilatation of the stomach veins.

The stomach is innervated by the vagus and sympathetic nerves. The pyloric part has a dense network of nerve fibres extending from both nerves. The larger curvature has a relatively sparse network of nerve fibres.

The stomach performs secretory, motor, absorptive and excretory functions.

I. Pavlov discovered the existence of a nervous regulation of digestion. He subdivided the digestive process into two phases: the conditioned reflex and the neuro-humoral. In the reflex phase, the gastric juice is secreted under the influence of neuropsychic stimuli such as the smell and appearance of food, layout of the table, which through the organs of sense are conveyed to the cerebral cortex. In response to them, a "conditioned reflex" juice is secreted in quantity even before eating. The secretion continues also after ingestion of food, under the influence of gustatory sensations derived from the food and the acts of chewing and swallowing. These propositions were proved by Pavlov and his followers by an experiment with a simulated feeding of a oesophagotomized dogs. In the second phase of gastric digestion, under the influence of the absorption of chemical constituents of food by the blood as well as secretion formed in the pyloric part of the stomach, gastric juice of a weaker digestive power and in quantities smaller than those seen in the conditioned-reflex phase is secreted.

The amount of gastric juice secreted in the stomach in 24 hours is 1.5-2 litres. Pepsin in an acid medium splits up proteins to the stage of albumoses and peptones; hydrochloric acid, acting upon animal and vegetable cellular tissue, prepares it for digestion in the large intestine. In addition, hydrochloric acid is an antiseptic agent killing microbes which in large numbers enter the stomach with food.

Mucus secreted by special cells protects the mucous coat of the stomach against mechanical and chemical irritation.

Pavlov established that both the quantity and quality of gastric juice in both phases of digestion are determined by the character of food. Fats inhibit gastric secretion.

Boiled vegetables, carbohydrates and alkaline water are mild irritants of gastric secretion. Meat, particularly roasted meat, tinned foods, ham, raw vegetables, mustard, pepper, salt and carbonaceous waters intensify gastric secretion.

Food entering the stomach undergoes the action of mechanical and thermal factors while it is moving from the cardia to the pylorus which relaxes and lets food pass into the duodenum. After this, the pylorus closes again, preventing the passage of new portions of food until the acid contents of the duodenum have been neutralized. The stomach is completely freed from ingested food in 3-6 hours, depending on the quality of the food. Meat and fats remain in the stomach the longest, whereas water and milk are the first to leave it.

The stomach absorbs alcohol, strychnine, sodium salicylate, etc. The absorption of saline and sugar solutions is negligible, while fats and water are not absorbed in the stomach at all.

Nitrogenous wastes accumulated in the body in large quantities in some diseases (uraemia, etc.), as well as dyes which had entered the body are secreted into the stomach by its pyloric part.

The stomach also secretes alcohol, morphine, and lactic acid injected parenterally.

Cardinal Symptoms of Stomach Disease

History taking. Very important for identifying a stomach disease are the patient's complaints and other anamnestic data. When questioning the patient, emphasis should be laid on the regularity of his diet, since irregular eating may cause a number of gastric ailments. Great importance is attached to the quantity and quality of ingested food (protein, carbohydrate, fatty, mixed, milk and vegetable) as well as overeating, undernourishment, and vitamin content of food. Note is taken of harmful dietary habits, such as hasty meals, eating dry food, and inadequate chewing of food which cause a mechanical irritation of the stomach, smoking, abuse of alcoholic drinks, eating pungent irritating food (spices, etc.). In collecting a case history, attention is paid to the patient's living conditions and environmental hazards. The patient's occupational surroundings are also taken into account because in certain cases working at a high temperature, in hot shops, or on jobs involving exposure to metal, coal, and cotton dust hazards may lead to a gastric disease. It is necessary to collect a history of past gastrointestinal diseases and operations performed on the digestive organs. For example, a history of a callous ulcer may point to the possibility of its malignant degeneration. An earlier operation for stomach cancer and complaints of functional disturbances in the digestive organs appearing several years after an operation may be suggestive of a cancer relapse.

Pains. In the presence of pains, attention should be paid to their localization, radiation, character, and association with the ingestion of food and the season of the year. Pains may be nagging, dull, lancinating, boring, and stabbing. They may be permanent or paroxysmal. The characteristics of pains may suggest the nature of the disease. For example, stabbing pains are felt in perforation of the stomach, pains felt late after the ingestion of food and hunger pain are commonly felt in duodenal ulcer, etc.

Disorders in the appetite. A considerable increase or reduction in the appetite is always a symptom of a certain disease. An impairment of the appetite (anorexia) is observed in case of an inflammatory process in the mucous membrane of the stomach or intestine, in stomach cancer, poisoning with toxic substances, etc., as well as in various types of nervous overstrain leading to an inhibition of the secretory function of the stomach.

An increased appetite is known to be concurrent in some cases with an increased stomach secretion, for example, in ulcer of the stomach or duodenum and in thyrotoxicosis. An excessive appetite (polyphagia), to the degree of morbid hunger (bulimia) is encountered in diabetes mellitus and after administration of large doses of insulin, cortisone, prednisolone, adrenocorticotropic hormone. A perverted appetite is found in nervous persons as well as in juvenile chlorosis when the sufferer willingly eats chalk, coal, lime. In rare cases a similar perversion of tastes is also encountered in pregnancy.

A partial and total loss of the sense of taste as well as an unpleasant taste in the mouth is often combined with coating of the tongue and may be encountered with a number of gastrointestinal diseases (gastritis, hepatitis, etc.).

Nausea and vomiting. Nausea and vomiting are found both with gastric and nongastric pathology. In nausea there is facial pallor, a decline in the arterial pressure, cold sweats and salivation. If the patient complains of nausea and vomiting, one should first of all consider a disease of the abdominal organs (gastric or duodenal ulcer, gastritis, alimentary intoxication, stomach cancer, helminthic infestation). Vomiting may be due to a conditioned reflex when a mere look at various excreta from the human body (sputum, faeces, etc.) or obnoxious odours provoke vomiting. An intense irritation of certain organs, such as the gallbladder, kidneys, vermiform appendage, heart, and peritoneum also causes vomiting. Nausea and vomiting are encountered in pregnancy and are sometimes a sign of toxaemia. Vomiting may be caused by irritation of the vomiting centre with various poisons circulating in the blood, as it takes place in azotaemic uraemia. Vomiting is noted in functional or organic disturbances of the central nervous system. In the presence of vomiting it is necessary to ascertain all circumstances responsible for it: the time of its onset, association with eating, food quality, connection with anxiety states. The character of the vomit and the presence of mucus and blood in it must be established.

Blood vomiting (haematemesis). It is encountered in diseases running their course with ulceration of the stomach mucosa (in ulcer, cancer, and polyposis), in ruptures of dilated vessels of the stomach and intestine, liver cirrhosis, thrombosis of the splenic veins, and in haemorrhagic vasculitis. In cases of profuse haemorrhage. the blood is bright-red, while when haemoglobin is exposed to the action of hydrochloric acid it takes the colour of coffee dregs.

In scanty and prolonged gastric haemorrhages the blood may be discovered by microscopy or chemical study.

Belching (eructation). This is due to the expulsion of air from the stomach into the oral cavity. Eructation may be empty (with air), or contain food. It may be acid as a result of fermentative processes, bitter due to the presence of bile, putrid or smell of a rotten egg when the air belched contains hydrogen sulphide formed in the stomach as a result of prolonged stagnation of its food contents due to an organic stricture or a functional spasm of the pylorus. A loud eructation is most commonly observed in persons with hysteria, neurasthenia, and after swallowing a considerable amount of air during a hasty meal (aerophagia).

Heartburn. Heartburn is a distressing burning sensation behind the sternum, particularly in its lower third and in the epigastrium owing to hydrochloric or organic acid entering the oesophagus from the stomach. Heartburn is found in persons abusing alcoholic drinks, spices, mustard, etc.

Thirst. Thirst is one of the signs of water deficit in the body. It is seen with a large blood loss, diarrhoea, as well as after copious repeated vomiting. Thirst in persons with endocrine and metabolic disorders (diabetes insipidus, diabetes mellitus) is not directly associated with the pathology of the digestive tract.

Impairment of Gastric Secretion and Evacuation

A disturbance in the secretory function of the stomach is manifested by an increase, a decrease or absence of gastric juice secretion.

Diminished gastric secretion (hyposecretio). Even under normal physiologic conditions the amount of free hydrochloric acid is subject to variations; it is sharply reduced during intense physical exertion. In pathology, a reduction in the secretory function of the stomach, to the degree of its total absence, is observed in febrile conditions, diseases of the spleen, liver, kidneys, etc. The absence of free hydrochloric acid is often noted in diseases accompanied by toxicosis and in a derangement of the function of the central nervous system causing inhibition of stomach secretion. The great importance of the central nervous system in the regulation of the secretory function of the stomach is illustrated by symptoms of heterochylia when the achylic condition of the stomach in a dejected person gives way to normal secretion when his mood becomes more cheerful. The loss of appetite in anxiety states and its increase in a cheerful, joyful mood also confirm the exceptional importance of the nervous system in the processes of juice secretion.

The secretion of free hydrochloric acid in case of impairment of the secretory function of the stomach is reduced (gastric hypoacidity or hypochlorhydria) or totally absent (anacidity or achlorhydria). Simultaneous absence from gastric juice of pepsin and hydrochloric acid is known as gastric achylia. Hydrochloric acid and pepsin may disappear as a result of organic or functional disturbances. The causes are differentiated by means of a histamine test. A thin tube is inserted into the stomach and 0.5 ml of a 0.1 per cent histamine solution is injected subcutaneously. In organic lesions of the stomach, hydrochloric acid is not secreted even after a histamine injection, and in case of a functional disorder free hydrochloric acid is discovered in the gastric contents. The secretory function of the gastric glands has long since been determined by means of an intramuscular injection of 2 ml of a 1 per cent neutral red solution with simultaneous insertion of a tube into the stomach. With a normal state of the glandular apparatus of the stomach the gastric contents take the stain in 12-15 minutes' time. The absence of stain for 40-50 minutes points to a complete atrophy of the gastric glands; a delay in staining over 20 minutes is noted in a reduction of their function.

Organic achylia of the stomach is encountered with atrophic processes in the gastric glands due to inflammatory changes, malnutrition, protein and vitamin deficiency in food—in scurvy, pellagra, beri-beri, anaemia, as well as with malignant newgrowths (cancer).

Increased gastric secretion (hypersecretio). An abnormal increase in the amount of gastric juice is known as hypersecretion, while an excessive acidity of gastric juice is known as gastric hyperacidity or hyperchlorhydria.

Hypersecretion is said to be present when, in the absence of obstruction of the pylorus, after Boas-Ewald's test breakfast the amount of aspirated gastric contents is in excess of 300 ml, and when more than 60 ml of gastric juice is extracted in one hour during examination with a thin tube.

As a rule, an increase in acidity occurs simultaneously with an increase in the amount of gastric juice although in certain cases the acidity of gastric juice may be normal or even reduced despite its excessive secretion. An increase in the secretion and acidity of gastric juice is observed in direct irritation of the gastric mucosa with food, in inflammatory changes of the gastric mucosa itself (gastritis, ulcer) or of the adjacent organs (liver, bile ducts, intestine, etc.), and in reflex stimulation of the mucosa via the vagus nerve. Hypersecretion and excessive acidity of gastric juice are identified from the patient's complaints of heartburn, acid eructation, sometimes vomiting, commonly encountered after eating fatty foods, rich and sweet dishes, and drinking wine, and are confirmed by laboratory examination. In some persons, gastric hypersecretion and hyperacidity cause no symptoms and are revealed accidentally during a prophylactic examination. Hypersecretion is sometimes manifested by a constant effusion of gastric juice containing free hydrochloric acid. In such cases, the amount of contents in a fasting stomach is in excess of 70 ml, and the patient complains of heartburn, vomiting, and hunger pains, i.e., the clinical picture resembles that of duodenal ulcer. Hypersecretion may also occur periodically, with a sudden copious and acid vomiting as a manifestation of a functional disturbance in the central nervous system. A classical example of periodic hypersecretion is gastric crises in tabes dorsalis.

Disorders of the evacuatory function of the stomach. Retardation of evacuation of the stomach contents is encountered in organic strictures of the pylorus and body of the stomach due to postulcerative scars, newgrowths, or a functional spasm. A reduction in the evacuatory function of the stomach is determined by withdrawal of a considerable amount of secreted material from a fasting stomach with remnants of food eaten the day before and a copious amount of gastric contents after a test breakfast. The evacuatory function of the stomach is more accurately determined by means of x-ray examination. The presence of barium in the stomach for 6 hours and longer is evidence of a considerable delay in evacuation. In a disturbance of the evacuatory function, food is propelled into the duodenum owing to hypertrophy of the pyloric muscles, although with a certain delay, but in time the stomach muscle loses its tone, becomes distended by the food contained in the stomach, the peristaltic movements of the musculature are weakened, and signs of gastric obstruction make their appearance. These disturbances are manifested by a periodic copious vomiting, sometimes extremely foetid, a feeling of heaviness in the epigastrium, and a considerable enlargement and ptosis of the stomach. In case of severe obstruction of the pylorus there is dehydration, a diminution in the urinary secretion and extreme emaciation of the body. Functional spasms of the pylorus never cause serious disturbances in the evacuatory function of the stomach and pass early under the influence of general invigorating treatment and atropine administrations.

Physical Methods for Examining the Digestive Organs

Examination of the oral cavity. The mucous membrane, the teeth, the gums, the tongue and the pharynx are examined. The oral mucosa, which is smooth and pink in healthy persons, may suffer a change in scurvy, mercury poisoning, uraemia, etc. In thrush, it is covered with white patches and plaques. Early after the onset of measles, the mucous membrane of the cheeks and lips shows white patches looking like pieces of lime, which were for the first time described by N. Filatov. In examining the teeth, attention is given to their number and condition. Carious teeth may become a source of various diseases. Often the form of the teeth may indicate the character of a disease. For example, in congenital syphilis there form semilunar notches on the free edges of the two upper incisors; the teeth are covered with rough transverse lines and are widely spaced. The condition of the gums may help identify lead poisoning, since the latter causes a bluish-black rim to form at the roots of the teeth.

In scurvy and mercury poisoning, the gums become spongy and bleed, and the teeth come out. This is also observed in diabetes mellitus.

Inspection of the tongue also gives valuable diagnostic evidence. The tongue may be covered with a white coating in diseases of the gastrointestinal tract and in fever. In scarlet fever the tongue is rough, bright-red in colour, with eminent papillae (raspberry tongue). A dry and foul-looking tongue with a thick coating in the centre and at the root is seen in enteric fever; in pernicious anaemia the tongue has a polished appearance as a result of atrophy of the papillae, the patient complaining of a burning sensation in the tongue, particularly when eating piquant foods.

The pharynx, the tonsils in particular, is inspected in broad daylight or with a bright electric illumination. Developmental anomalies are detected, such as wolf jaws, i.e., a high and cleft palate communicating with the oral and nasal cavities. For examination of the pharynx, the patient is enjoined to utter the sound "a-a-a" or "e-e-e" while the root of the tongue is pressed with a spatula. Succulent, spongy and enlarged tonsils are observed in chronic inflammations. Tonsils of a red colour, with a white film consisting of lustrous spots, are encountered with follicular tonsillitis. A greyish membrane firmly attached to the tonsils and extending to the arches and uvula is highly pathognomonic of diphtheria. Swelling of one of the tonsils causing an intense pain on swallowing may be the result of a peritonsillar abscess.

In examination of the oral cavity, various odours emitted from the latter should be determined.

Inspection and light palpation of the abdomen. The abdomen is examined in a standing and recumbent posture of the patient. Attention must be given to the form and size of the abdomen, abnormal bulgings, a visible intestinal peristalsis, as well as the participation of the abdominal wall in the respiratory act. The abdomen of a normal configuration must be on a level with the chest both in the vertical and horizontal positions. An enlarged abdomen is observed in obesity, pregnancy, meteorism, collection of fluid in the abdominal cavity, and with large tumours in the abdominal cavity and organs of the small pelvis. In some cases enlargement of the abdomen may be regular (for example, in obesity, meteorism); in others, irregular (for example, in ascitis).

Tumours sometimes produce a partial enlargement of the abdomen corresponding to the tumour site. In visceroptosis the upper part of the abdomen is indrawn, the lower one is protruding.

In inspection of the abdomen, it is necessary to determine a possible diastasis of the rectus muscles as well as the presence of hernias: umbilical, inguinal and epigastric. Attention is also paid to the vascular system of the abdominal wall. Normally, the vessels are not visible; in stasis, in the portal vein, however, collateral circulation occurs through the superficial veins of the anterior and lateral parts of the abdomen, which causes the formation of the so-called caput medusae.

Determination of visible peristalsis may sometimes, after light strokes, give valuable evidence of the changes occurring in the gastrointestinal tract. Striking the region of the stomach with half-bent fingers (succussion) in a fasting patient with a stenosed pylorus may elicit a splashing sound, which points to retention of food in the stomach. In addition, in pyloric stenosis an intense peristalsis is visible to the naked eye. This peristalsis begins in the epigastrium and is directed from above downwards and to the right, while antiperistalsis involves contraction of the stomach in the opposite direction. In intestinal obstruction due to various causes, an intense intestinal peristalsis is visible above the site of obstruction.

It is important to determine the movements of the abdominal wall during respiration in view of the fact that in inflammation of the peritoneum the abdomen takes no part in respiration, whereas normally it does so simultaneously with the thorax.

A freely displaceable collection of fluid in the abdominal cavity is seen in a number of diseases. In the presence of general oedema, mechanical compressions and stasis in the vessels of the abdominal cavity there is a collection of non-inflammatory fluid (transudate); the accumulation of fluid in the abdominal cavity is termed ascites. During inflammatory processes in the peritoneum and the abdominal organs as well as in case of tumours of the peritoneum an inflammatory fluid (exudate) accumulates in the abdominal cavity.

Determination of a large collection of fluid in the abdominal cavity is not difficult. The external aspect of the abdomen is highly characteristic: in a standing posture it is prominent and somewhat sagging, sometimes with a protruding navel; in a recumbent posture it spreads on the sides to form the so-called frog belly. The skin over the abdomen is lustrous, stretched, smooth and thin. The presence of fluid in the abdominal cavity may be determined by percussion and palpation. Percussion of the abdomen in a healthy person elicits a tympanitic resonance due to the presence of gas in the intestine and stomach. In ascites the fluid fills the lower parts of the abdomen when the patient is standing, while the intestinal loops are raised. Therefore tympanitic resonance is heard over the umbilical region, whereas below the navel percussion will elicit a dull sound. When the patient is lying on the right side, the fluid will be displaced to the same side by force of gravity, the dull sound will be audible at right, and the tympanitic resonance, at left. A reverse picture is obtained when the patient is lying on his left side. The presence of fluid in the abdominal cavity is determined by palpation or from fluctuation when the patient is in a recumbent posture. The fingers of the left hand are applied to the right side of the patient's abdomen, while the right hand is used to make light pushing strokes over the left side (Fig. 52). The presence of fluid will give rise to waves, whose movement will be transmitted to the right side and will be felt by the fingers as a push. The latter, however, may be caused by fluctuations of the abdominal wall. To rule out this error, assistance is required from another person, who is told to place his hand (or book) edgewise on the abdominal wall with a slight pressure.

The most reliable method for determining fluid in the abdominal cavity is puncture of the latter with a special needle known as trocar. A puncture serves to find out not only the presence but also the character of fluid (transudate, haemorrhagic, serous, purulent and other exudates). In the fluid obtained one should determine the specific gravity, protein level, and cellular composition of the sediment in which erythrocytes, leucocytes, cells of the peritoneal endothelium, and sometimes cells of newgrowths may be discovered.

For a puncture of the abdominal cavity with a trocar, the patient is at first told to evacuate the urinary bladder, after which he is seated on a chair or on the edge of a bed. The puncture is made at a point between the navel and pubis in the midline or 2 cm to the side from it. The site of puncture and the adjoining region are wiped with alcohol and painted with iodine tincture. A trocar preliminarily sterilized is introduced into the abdominal cavity by rotary movements, after which the stylet is drawn out from the cannula.

Ascitic fluid escapes through the trocar cannula in a strong jet. As the flow of fluid grows weaker the abdomen is gradually tightened with a broad towel preliminarily placed over it. Some 10-12 litres of fluid may escape at a time, after which the cannula is withdrawn, and the site of puncture is covered with a sterile square of cloth, which is fixed in place with an adhesive plaster or iodoform collodium (Fig. 53).

Palpation of the Abdomen

Palpation of the abdomen serves to furnish important information on the location of the organs of the abdominal cavity, changes in their form, consistency, sensitivity, motility, etc. During palpation the patient must breathe deeply, relaxing his abdominal muscles. The examiner's hands, which must be warmed, are freely placed on the abdomen. In order to relax the abdominal muscles, it is recommended to distract the patient's attention. Palpation may be performed in a recumbent and standing posture of the patient. In the recumbent posture, the patient's arms lie along his sides, while his head is rested on a low pillow. The examiner is seated on his right. Palpation may be light or deep.

Light palpation. For light palpation, the examiner lays his hand flatwise on the abdomen and inspects its entire surface with light touches. This serves to ascertain the character of tension of the abdominal wall: whether it is local or general, due to ascites or

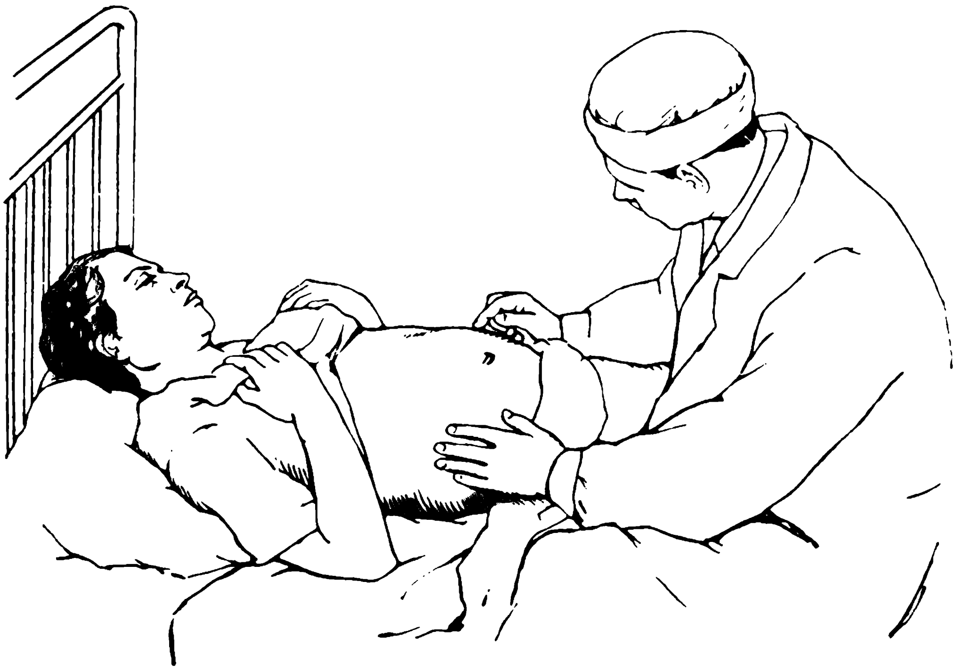

Fig. 52. Examination of fluctuation in ascites

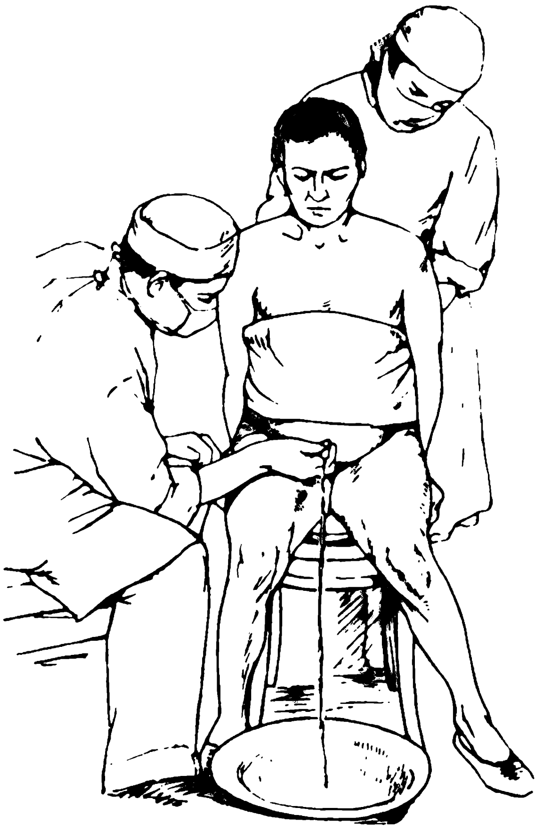

Fig. 53. Puncture of the abdominal cavity in ascites

contraction of the abdominal muscles resulting from peritonitis (general tension) or a local irritation of the peritoneum in appendicitis (right inguinal region), caused by gastric or duodenal ulcer (epigastrium) or acute and chronic cholecystitis (right hypochondrium). The tension of the muscles of the abdominal wall is called the muscular defence.

Light palpation also reveals isolated sites of pain. General tenderness over the entire abdomen is felt in peritonitis after perforation of an ulcer, the vermiform appendage, etc. Pain in the epigastrium is felt in gastric ulcer. Tenderness at the site of intersection of the 10th rib with the rectus abdominis muscle at right is encountered with an inflammatory process in the gallbladder. A painful point in the right lower quadrant of the abdomen is highly characteristic of an inflammation of the vermiform appendage. Tenderness may be not only punctate in character but it may also spread over the entire region occupied by an organ, as is the case in diseases of the stomach, the liver, the spleen, etc.

Palpation helps detect *epigastric*, *inguinal* and *umbilical* hernias. Epigastric hernia is best to detect when the patient is standing. The examiner carries his finger from the xiphoid process to the navel, and if a hernia is present, the finger will feel a bulge. In doubtful cases, the patient is told to strain himself or cough to make the hernia more palpable. Umbilical hernia, as a rule, forms a bulge in the umbilical orifice. Small umbilical hernias are detected by palpation when the patient is coughing, which makes their presence quite obvious. Inguinal hernia results from distention of the inguinal orifice, which is normally as narrow as to let in only the tip of the little finger. The hernia is detected by insertion of a finger into the inguinal orifice.

Methodic deep, sliding palpation. The best way to determine changes in the organs located within the abdominal cavity is a methodic deep, sliding palpation suggested by V. Obraztsov (1886). It essentially consists in a cautious and gradual pressure on the abdomen with four half-bent fingers during each inspiration to reach the posterior abdominal wall. The organs palpated—the stomach, intestine, kidneys, etc.—are pressed with the fingers to the posterior wall; during an expiration, they slip out from under the fingers to give the examiner an idea of their size, consistency and pathologic changes.

The abdominal organs are palpated in the following sequence: the sigmoid colon, the caecum, the vermiform appendage, the transverse colon, the stomach, the liver, the gallbladder, the spleen, the kidneys, and the rectum.

The sigmoid (*pelvic*) *colon* (S-romanum) is located in the left iliac region, is provided with a long mesentery, and therefore its position is subject to variations depending on the latter's length. Palpation of the sigmoid colon is performed as follows (Fig. 54):

The hand with its four fingers slightly bent is laid on the left iliac region, the skin is slightly drawn to the midline, and the fingers are slowly depressed until they reach the posterior wall of the iliac fossa, after which they slip over it together with the skin. In healthy lean persons, the sigmoid colon is nearly always palpable as a cylinder of dense consistency, with smooth walls as thick as a thumb. Its palpation is painless and does not cause rumbling in the bowels (borborygmus). It is displaceable to both sides within limits of 3-5 cm. An irregular and rosary-like form of the intestine is noted in case of an accumulation of compact faecal matter or faecal stones within it. An excessively dense consistency and knobbiness of the sigmoid colon are seen with tumours, while rumbling and splashing sounds are heard in diarrhoea or when fluid and gas accumulate within the intestine. Inflammatory changes in the sigmoid colon are accompanied by its spastic contractions and marked tenderness on palpation. Sometimes, it may be fixed by adhesions.

The caecum lies in the right iliac region. For its examination, the tips of slightly bent fingers are laid on the right iliac region, slowly pressed down to the posterior wall and slipped across the caecum. Normally, the latter is palpable as a 2-3 cm thick elastic cylinder, giving out a low rumbling sound but painless and limited in mobility because of the short mesentery. A knobby dense formation palpable in this region is a very characteristic sign of tuberculosis and cancer of the caecum. In appendicitis, an inflammatory infiltrate may sometimes be felt near its site. An inflamed caecum is painful, begins to give out rumbling sounds, while its mobility is even more limited by adhesions. With an elongated mesentery, the caecum may become readily displaceable and is known as a mobile caecum.

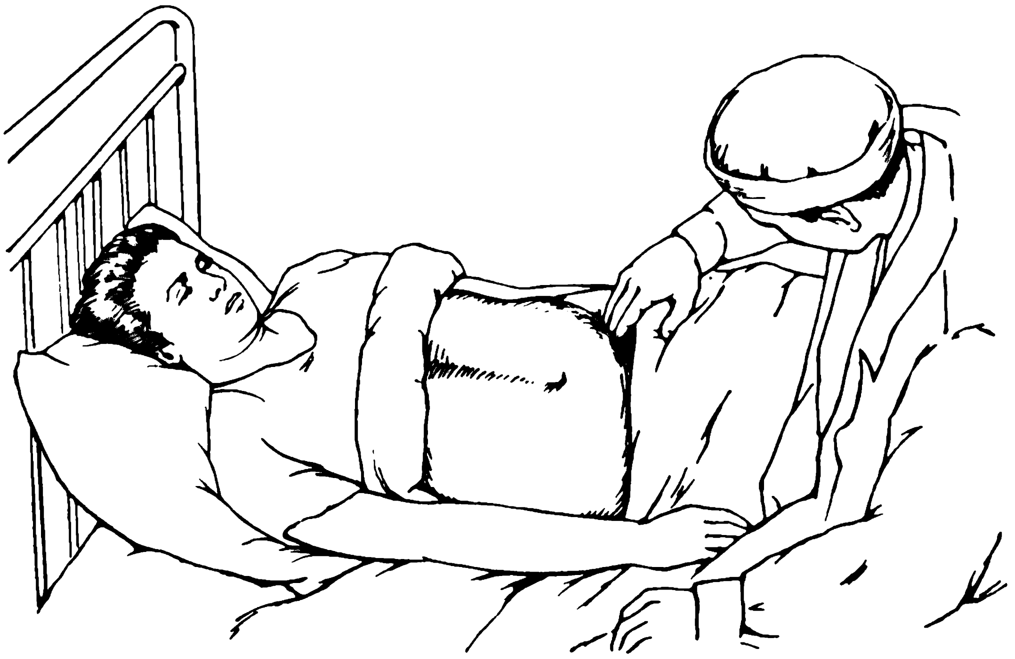

Fig. 54. Palpation of the sigmoid colon

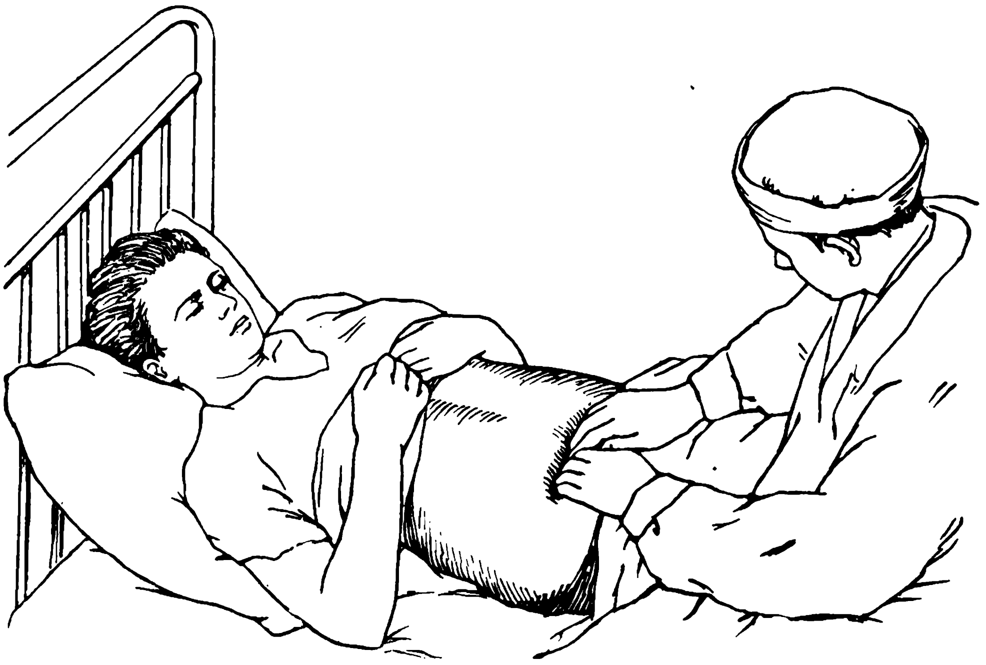

Fig. 55. Palpation of the transverse colon

The vermiform appendage (appendix) branches off from the posteromedial aspect of the caecum, lies medially to it and descends downwards. Sometimes it is situated behind the caecum. Normally the vermiform appendage is palpable with great difficulty. It is readily palpable in inflammations, when it is thickened, limited in mobility and painful.

The transverse colon (colon transversum) is located transversely, above the level of the navel. It is palpated below the stomach by one or both hands (Fig. 55). Even under normal conditions its position is variable: it is either located transversely, passing from the hepatic angle to the splenic angle, or insignificantly sags in the centre, and is sometimes entirely located within the small pelvis. An empty transverse colon is palpable as a transverse band. In the presence of gases it is felt as a soft body, whereas dense faecal matter lends it the consistency of a solid body. In the presence of a tumour, the transverse colon becomes excessively dense in consistency, knobby and limited in mobility. In case of stricture in any of its parts the colon is distended above the site of constriction and sometimes shows a vigorous peristalsis.

In *palpation of the rectum* the patient assumes a knee-and-elbow position, i.e., he rests on his knees and elbows. Sometimes palpation is performed in a lateral position of the patient. In this case, he lies on his side, his legs being drawn to the abdomen. The examination is begun with a local inspection of the perineum, for which purpose the buttocks are drawn apart with the hands. This may expose external haemorrhoids, fissures, fistulas, and redness of the skin. This is followed by palpation of the rectum with an index finger greased with fat or petrolatum oil, which is inserted into the anus as far as possible.

ACUTE GASTRITIS (GASTRITIS ACUTA)

Gastritis is an inflammation of all the three coats of the stomach, i.e., the mucous membrane, the musculature, and the serous membrane. In respect of its clinical course, gastritis may be acute or chronic. Acute gastritis may, in its turn, be subdivided into exogenous or simple, haematogenous, corrosive or toxic, and phlegmonous.

Aetiology and pathogenesis. Acute gastritis is due to chemical and thermal factors irritating the stomach through the agency of food and blood. The most frequent causes are abundant meals, hardly digestible, fatty, piquant, too hot or too cold food, strong liquors, and spoiled foodstuffs. The condition may also be caused by hypersensitivity to a number of medicinal substances (digitalis, iodine, bromide, sulphonamides, biomycin, terramycin, etc.) and to certain foods (eggs, strawberries, milk, shellfish, etc.).

Gastritis may develop with a number of food poisoning (salmonellosis, botulism, etc.). An important role in the origination of gastritis is played by past diseases, neuropsychic disturbances, fatigue, etc.

Pathologic anatomy. In simple gastritis the mucous membrane of the stomach is diffusely affected, but the region of the pylorus is damaged to a greater extent. The gastric mucosa is congested and oedematous, covered with mucus and leucocytes, while its secretion is reduced; small areas of bleeding and erosion may be encountered. The interstitial tissue is infiltrated by leucocytes.

Clinical picture. An acute inflammation of the gastric mucosa often runs its course with negligible symptoms of dyspepsia and sometimes with several local and general changes. Usually gastritis develops suddenly, in 6-12 hours after a meal or later. The cardinal clinical symptoms include loss of appetite, an unpleasant taste in the mouth, salivation, nausea and repeated vomiting, a feeling of fulness and heaviness in the epigastrium, and slight pains in the stomach region. The vomit contains mucus, bile, rarely an admixture of blood, and undigested food particles, and emits an obnoxious acid odour. In repeated attacks of vomiting, expulsion of food material from the stomach is followed by a discharge of a sticky mucus stained with bile. A recollection of food causes attacks of vomiting. The patient feels indisposed, is very thirsty because of a large loss of liquid with the vomit, complains of dizziness and headache. The body temperature may be subfebrile, sometimes febrile, following a chill and herpes labialis. The patient is pale, the pulse is quickened, and the tongue is covered with a greyish-yellow coating. Palpation of the stomach region causes a slight pain, and nausea becomes worse on pressure applied to the epigastrium. The urinary secretion is diminished, and protein is sometimes found in the urine. The blood shows a picture of neutrophilic leucocytosis. Gastric secretion, which is at first increased, later decreases even to complete disappearance of free hydrochloric acid. The evacuation of the stomach contents is delayed, while the absorptive capacity of the mucous membrane is elevated.

The relatively grave condition lasts not longer than 1-3 days. Upon recovery, acute gastritis often recurs under the influence of the same factors. Sometimes the gallbladder is also inflamed in acute gastritis.

Acute gastritis must be differentiated from a number of diseases producing a similar clinical picture, such as appendicitis, myocardial infarction, cholecystitis, gastric and duodenal ulcer, and intestinal obstruction, as well as from acute diseases accompanied by vomiting (meningitis, enteric fever, paratyphoid, scarlet fever, etc.).

Treatment and prophylaxis. Prophylaxis consists in measures to eliminate the factors responsible for acute gastritis and in institution of a healthful diet. First of all, stomach lavage should be performed to remove the food and toxic substances which have caused gastritis. For this purpose, the patient is seated on a chair, a tube is inserted into his mouth with the right hand and, while the patient swallows, it is slowly pushed down over a length of 40 cm corresponding to the distance from the teeth to the stomach orifice. The patient is told to strain himself, and the gastric contents are discharged through the tube into a funnel attached to it, and poured out. The funnel is then filled with 1-2 litres of warm boiled water with a 2 per cent soda solution, raised high to let the water enter the stomach and then quickly lowered to the level of the stomach.

According to the law of communicating vessels, the water flows from the stomach into the funnel. This procedure is continued until the lavage water has been freed from remnants of food and mucus, and has become transparent. When it is impossible to lavage the stomach by means of a tube, the patient is given several glassfuls of warm mineral alkaline water, which contributes to the cleansing of the stomach from mucus and poisonous substances by repeated vomiting.

In cases where lavage cannot be made by conventional methods, apomorphine (formula No. 84) is injected subcutaneously, and purgatives are given when the clinical picture is joined by enteritis.

The patient is confined to bed, and heat is applied to the abdomen to relieve pains; in severe debility and collapse his feet are warmed by heaters, and camphor and caffeine are injected subcutaneously. In severe dehydration of the body subcutaneous or intravenous injections of physiologic salt solution or a 5 per cent glucose solution in quantities of 0.5-1 litre are recommended. During the first two days the patient is advised to abstain from food and is given liquid to drink in small portions (cool tea, mineral water). In case of nausea, he is given pieces of lemon and ice to suck. On the 2nd-3rd day liquid food is prescribed: tea, coffee, sour milk, kefir, lean broth, mucilaginous soups, later thin gruels, jelly, purée, puréed meat, soft-boiled eggs, and rusks. The variety of food given is widened gradually and within 7 to 10 days the patient is transferred to an ordinary diet.

In allergic gastritis it is recommended to administer adrenalin subcutaneously, calcium chloride per os, calcium gluconate intravenously, and dimedrol (formula No. 173), while intakes of food responsible for the allergic reaction are forbidden.

It is necessary to keep to a strict routine in the storage of foodstuffs, particularly in summer when food readily gets rotten. Foodstuffs bought on a market should be washed carefully, boiled, cooked, and stored in a cold place, and meals should be preferably cooked for one day. Kitchen utensils should be kept clean. It is recommended to carry out strict hygienic examination of personnel of restaurants and dining-rooms and shop assistants in all food stores.

Acute haematogenous gastritis occurs with a variety of diseases, especially often with infections (enteric fever, influenza, dysentery, scarlet fever, etc.). It is due to accumulation in the blood of decomposition products of the patient's own proteins in burns, fever, thyrotoxicosis, hepatitis, and on exposure to the action of x-rays and ultraviolet light.

Another haematogenous disease is azotaemic gastritis in which the gastric mucosa is affected by toxins excreted by the stomach in uraemia.

The patient complains of a loss of appetite and sometimes of persistent vomiting. In the presence of the above diseases, the food must be bland in order to avoid irritation of the stomach not only during an acute process but also in convalescence so as to forestall the transition of an acute gastric inflammation into a chronic form.

Phlegmonous gastritis most commonly develops as a secondary condition in enteric fever and as a complication of gastric ulcer or gastric cancer. It is prevalent in men, particularly in alcoholics. The causal agent is a streptococcus, staphylococcus or pneumococcus, which produces a diffuse suppuration of the stomach wall.

The disease is acute in onset, with a chill and fever of up to 40°C, with pains in the epigastrium. The patient's condition rapidly deteriorates, with repeated chills and frequent vomiting at first of food material and later of bile, pus sometimes being found in the vomit. There is diarrhoea and intense thirst. The tongue is dry, the abdomen is inflated, and rigidity of the abdominal muscles, marked tenderness and sometimes a painful tumour in the epigastrium are revealed on palpation. The pulse is rapid and small. The patient dies in a state of collapse from sepsis or peritonitis. The disease is rarely identified during life, and is usually mistaken for acute cholecystitis, acute pancreatitis, or an intestinal abscess.

Acute corrosive gastritis results from the entry into the stomach of caustic alkalis, concentrated acids, corrosive sublimate, arsenic, and other substances, which cause an intense irritation and destruction of both the oesophagus and the mucosa and other membranes of the stomach. Pronounced hyperaemia, oedema, haemorrhages, superficial or deep ulcers of the mucous membrane, and even perforation of the stomach may be encountered. The degree of damage to the stomach depends on the character and concentration of poison as well as on the food contents of the stomach at the time of poisoning.

Immediately upon ingestion the poison causes an intense burning pain in the mouth, the oesophagus and the epigastrium. There is vomiting with a discharge of mucus, blood, remnants of food, and scraps of the oesophageal or gastric mucosa. Traces of a burn are found on the lips, and on the mucous membranes of the mouth and pharynx. Oedema of the larynx develops and the voice becomes hoarse or is lost completely. In grave cases, death follows within the first hours or days in a state of shock or from peritonitis. In mild cases ending in recovery, scars form in the oesophagus and stomach, which narrow the oesophagus and pylorus. In addition, symptoms of chronic gastritis make their appearance.

A correct treatment given in due time brings down the mortality rate and improves the prognosis. It is necessary, first of all, to remove the poison by means of lavage of the stomach with warm water through a large-diameter tube. The insertion of a tube during shock is contraindicated. Antidotes are also prescribed: milk, lime water, milk of magnesia for poisoning with acids; dilute citric or acetic acid for poisoning with alkalis. In a state of shock, repeated injections of caffeine, cordiamine, sympathol, and transfusions of blood substitutes, plasma and blood are necessary. To relieve pain narcotics (morphine, pantopon, etc.) are administered. In the early days of the disease, subcutaneous injections of physiologic salt solution, or a 5 per cent glucose solution introduced by intravenous drip or an enema are recommended. Bouginage is undertaken to prevent strictures of the oesophagus during healing. Perforation of the stomach requires surgical treatment.

CHRONIC GASTRITIS (GASTRITIS CHRONICA)

Numerous external and internal factors are known to have a role in the genesis of chronic gastritis: eating coarse, spicy foods, drinking alcoholic beverages, irregular meals, overeating, inadequate chewing of food due to defects in the masticatory apparatus, a low caloric, vitamin and protein value of food, etc. Certain irritants of the gastric mucosa and occupations involving exposure to metal, cotton, coal, and silicate dusts, and to alkali and acids also contribute to the development of chronic gastritis. The secretory function of the stomach is also adversely affected by working in hot surroundings, and in the meat-packing industry, where minced meat is often eaten raw by factory personnel. A similar role may sometimes be played by certain medicines (salicylic drugs, diuretin, soda, purgatives) administered to hypersensitive subjects. Chronic gastritis may also be due to the influence of certain acute (fevers, influenza, enterocolitis) and chronic (malaria, syphilis, tuberculosis, etc.) infections and local septic foci (tonsillitis, stomatitis, cholecystitis, etc.). Other contributing factors in the aetiology of chronic gastritis are diseases of the liver, the kidneys, and the haemopoietic system, diabetes, obesity when, owing to an impairment of the barrier function of the liver, toxic substances produce a harmful influence on the stomach by way of the blood stream. Sometimes, the development of gastritis is associated with a circulatory disturbance in the stomach and oxygen deficiency, which is seen in cardiovascular insufficiency. Chronic gastritis may result from functional disturbances in the stomach, in particular in prolonged stimulation of the vagus nerve resulting in a copious secretion in an empty stomach, which is associated with chronic nicotine intoxication. Chronic gastritis may in certain cases be secondary to untreated acute gastritis.

The former division of gastritis into the atrophic and hypertrophic forms, and the latter, into hyperacid and normally acidic forms cannot be considered convenient. In atrophic gastritis, areas of proliferation of the mucous membrane are seen simultaneously with areas of atrophy, while in hypertrophic gastritis there are also areas of atrophy. A distinction of forms of gastritis for the degree of gastric juice acidity is also incorrect because one symptom, even if cardinal, is not enough for revealing the essence of a disease. In view of these considerations, it is best to subdivide all forms of gastritis into two large groups: (a) gastritis with secretory insufficiency, with a reduced secretion or absent free hydrochloric acid; (b) gastritis with an increased or normal secretion.

Gastritis with secretory insufficiency. An important sign of this variety of gastritis is a reduction in or absence of free hydrochloric acid. If no pepsin is discovered in the gastric juice in the absence of free hydrochloric acid, the condition is known as gastric achylia. The latter, however, may be organic and functional in origin. These two forms are differentiated by means of subcutaneous injections of histamine or neutral red.

The term "gastritis with secretory insufficiency" implies an organic inflammation of the stomach running its course with a reduction to the degree of complete suppression of the secretory, enzymatic, and the evacuative functions of the stomach.

Clinical picture. Gastritis with secretory insufficiency is the most frequent in middle-aged and elderly persons. In the early period of the disease the patient feels no discomfort because the digestive function is taken over by the compensatory mechanisms of the pancreas and the small intestine. In time a disorder in digestion becomes apparent. It is manifested by a sensation of burning, fullness, and pains in the epigastrium, frequent loud belching of air, at times with a rotten egg or acid taste, the latter apparently due to the formation of organic acids—acetic, lactic, and butyric—as a result of fermentative processes occurring in the stomach. The patient vomits frequently, particularly in periods of exacerbation. In alcoholism, a characteristic feature is morning sickness (vomitus matutinus) which is partly accounted for by concomitant pharyngitis and laryngitis. Pains are infrequent and are commonly felt by persons with a simultaneous affection of the intestine and the bile ducts.

Gastritis with secretory insufficiency may be attended with a disturbance in intestinal digestion and with diarrhoea (gastrogenous diarrhoea). The latter is due to the loss of bactericidal properties by the gastric juice and a deficit or absence of pepsin, as a result of which digestion of connective tissue of animal or vegetable origin becomes inadequate. Because of this, the inadequately digested food rich in bacterial flora, upon its entry into the small intestine, either irritates the latter or causes inflammations entailing diarrhoea of a fermentative or putrefactive character. Diarrhoea is also likely to appear where chronic gastritis is complicated by an infectious process developing in patients after ingestion of spoiled food. Constipation is also noted sometimes.

Changes in the intestine are at first functional in origin, and later are joined by inflammatory changes as evidenced by stomach pains and the presence of mucus and leucocytes in the faeces. The faecal matter contains undigested muscle fibres with nuclei and a considerable amount of vegetable cellular tissue. All this causes emaciation, hypoproteinaemia, endogenous vitamin deficiency, and anaemia. Emaciation is sometimes due to abstinence from food for fear of pains, while polyavitaminosis is consequent upon a prolonged lenient diet deficient in vitamins and administration of sulpha drugs which destroy vitamins of the B complex. Many vitamins are eliminated with liquid faecal matter, while vitamin C is destroyed in the alkaline medium of the stomach. Hypo- and avitaminosis is manifested by general debility, early fatigue on exertion, spongy and bleeding gums, dry skin, and glossitis. Pellagra is a rare occurrence but sometimes there are mild symptoms of this disease such as isolated areas of a grey-white colour, or a pigment circle around the anus and on the buttocks and elbows.

In gastritis with secretory insufficiency changes occur in the liver, pancreas, haemopoietic system, and the neuropsychic sphere.

The liver and bile ducts are affected by ascending infection which extends from the intestine or through the portal vein. Concurrent gastritis may also be secondary to an impairment of the barrier function of the liver as is the case in Botkin's disease (infectious hepatitis) and angiocholecystitis. The pancreas is also involved in the disease process. As is known, a 0.5 per cent hydrochloric acid solution is a physiologic stimulant of the pancreas, and it would be natural to expect a sharp impairment of the latter's function in secretory insufficiency of the stomach. However, diluted vegetable juices and bile also stimulate pancreatic secretion, owing to which the pancreas is for a long time able to compensate for the diminished function of the stomach. A reduction in pancreatic function is seen with a simultaneous affection of the stomach and liver.

The haemopoietic system shows a variety of disturbances. If gastritis is localized in the fundal part hyperchromic anaemia develops in connection with the death of the cells producing gastromucoprotein. The latter combines with vitamin B_{12} from the food to safeguard it against destruction by the intestinal bacterial flora. After being absorbed through the intestinal wall, vitamin B_{12} is freed of gastromucoprotein and combines with α-globulin to form vitamin B_{12}-complex which is deposited in the liver, muscles and other organs. Hypochromic iron-deficiency anaemia develops when the gastric juice contains no hydrochloric acid which converts ferric oxide into ferrous oxide. In gastritis with secretory insufficiency, the patient has poor tolerance of certain foods: eggs, fish, berries, etc. This is accounted for by the fact that incompletely split-up products, being absorbed by the blood, cause symptoms of hypersensitivity manifested by urticaria, Quincke's oedema, etc.

The disease runs a characteristic course with periodic remissions after treatment followed by relapses due to a fault in the diet anxiety, infection and chilling. The diagnosis is based on clinical, laboratory, x-ray and gastroscopic studies. Gastroscopy is sometimes poorly tolerated by the patient. A gastroscope can be used to determine various forms of gastritis, study the changes occurring during the course of the disease and detect a new growth or an ulcer. Gastroscopy is contraindicated in the presence of aortal aneurysms, hypertensive vascular disease, strictures of the oesophagus, and inflammatory changes in the upper air passages.

In chronic gastritis with secretory insufficiency atrophy of the mucosal folds is seen together with areas of hypertrophy, not only narrow but also normal and even wide folds of the mucous membrane being determinable. The gastric tone is diminished, while the evacuative function is unchanged. X-ray examination plays no substantial role in the diagnosis of gastritis.

Laboratory examination of gastric juice reveals highly characteristic features of gastric contents obtained after Boas-Ewald's test breakfast. The bread seems to have been chewed and spit out, while the liquid layer is transparent and nonopalescent, the correlation between the solid and liquid parts being 1 : 1 or 1 : 2 instead of 1 : 3. The contents are stained with bile and have a putrid odour. The presence of lactic acid and Boas-Oppler bacilli are observed in stagnation of the gastric contents, with a tumour at the exit of the stomach. Total acidity is within limits of 6-10 units, free hydrochloric acid is absent or reduced in content. The digestive capacity of pepsin is considerably reduced or absent. Microscopy reveals a considerable number of epithelial cells, leucocytes, and mucus.

Gastritis with secretory insufficiency may give rise to gastric polyposis or gastric carcinoma. Therefore in each case gastritis with secretory insufficiency should be differentiated from these conditions.

Polypous gastritis or gastric polyposis is a rather frequent disease. Polyps belong to the group of benign tumours and consist of a connective-tissue stroma and tegumentary (papillomas) or grandular epithelium (adenomas). Polyps may be soft or hard, mostly multiple, attached by a pedicle or by a broad base, and resemble berries or cauliflower in appearance. Their diameter is rarely in excess of 5 cm, and is more commonly within limits of 1-2 cm. They are localized most frequently in the antral part, on its anterior or posterior wall. Gastric polyposis should be regarded as a complication of gastritis with secretory insufficiency. It is mainly encountered at ages of 40 to 50, less often at a younger age. The patient complains of dull epigastric pains on ingestion of food, belching of air, sometimes with a putrid odour, heartburn, nausea, and, less frequently, vomiting. In the majority of cases, gastric juice acidity is totally absent or reduced. There is predisposition to scanty bleeding, while profuse haemorrhage occurs in strangulation of polyps in the pylorus, in their prolapse into the duodenum, as well as in cancerous degeneration.

X-ray examination is of decisive importance for diagnosis. Polyps produce filling defects with regular contours, which are readily displaceable (if attached by a pedicle). Mucosal folds at the site of a polyp do not break off as distinct from those in cancer, and do not converge radially, like those in ulcer, but form a bypassing arch. It is highly important to decide whether a cancerous degeneration of the polyp has occurred. X-rays are of little help in such cases. Evidence in favour of cancerous degeneration is progressive anaemia, loss of flesh, and other signs characteristic of cancer.

Polypous gastritis should be differentiated also from gastric cancer, which may be a primary disease while gastritis may be secondary. Sometimes chronic gastritis with secretory insufficiency is complicated by a concurrent stomach carcinoma. In such cases the existing signs of gastritis are joined by new symptoms, for example, stagnation of food in the stomach, occult blood in the faeces, a positive reaction to lactic acid, and the presence of Boas-Oppler bacilli in the gastric juice. Cytologic examination of lavage water from the stomach reveals desquamated cancer cells. Severe emaciation, fever, and a palpable tumour appear only at later stages of malignancy. An important role in the diagnosis is played by x-ray and gastroscopic examination.

Prophylaxis consists in observance of the rules of personal hygiene and healthful dietary habits. Carious teeth must be treated and missing teeth replaced by dentures. It is necessary to give up smoking and abstain from alcohol. Food must be taken frequently but in small portions and well chewed. It should be nutritious and bland, eaten minced or puréed, and sufficient in animal proteins and vitamins of the B and C groups. It is allowed to eat certain condiments stimulating the secretion of gastric juice, and to drink mineral water which contains common salt and increases secretion. Mineral water is drunk warm by mouthfuls half-an-hour before meals. Very helpful are periodic lavages of stomach with a 2 per cent soda solution dissolving mucus, or with alkaline mineral water. Hydrochloric acid with pepsin, acidol pepsin (containing 27 per cent of hydrochloric acid), natural gastric juice producing a bactericidal effect, pancreatin (formula No. 75), as well as various bitters (formulas Nos. 73, 74 and 74*a*) intensifying gastric secretion are administered per os. Sanatorial and health resort therapy is also advisable.

Gastritis with retained and increased secretion. This disease affects young and middle-aged persons. Exacerbations occur in spring and autumn due to dietary faults. Disorders in the neuropsychic sphere are also among the contributing factors. Sometimes gastritis with a normal or increased secretion gives rise to gastric and duodenal ulcer.

Chronic gastritis with a retained and increased secretion may be subdivided into two forms: dyspeptic and painful.

A patient with a *dyspeptic form* complains of acid belching, regurgitation of acid matter, heartburn, a burning sensation in the epigastrium, and pressure and distention of the abdomen. Vomiting is very rare. The appetite is retained and even increased. Its absence is most commonly due to a fear of eating. These symptoms are often of paroxysmal character, appear after breakfast, or dinner and pass quickly after administration of soda. Sometimes a patient is discomforted only by heartburn or a feeling of heaviness after a meal or after a violation of the diet, i.e., after eating fatty foods, ingestion of a considerable quantity of carbohydrates, alcohol, etc. These symptoms are due to a disturbance in the secretory function of the stomach as well as to hypersensitivity of the gastric mucosa.

A patient with a *painful form* feels late hunger pains, sometimes night pains, which subside after a meal or an administration of soda. They vary in intensity and may be dull or nagging, and are localized in the epigastrium. Often, pains occur in combination with signs characteristic of the dyspeptic form of gastritis (heartburn, flatulence, etc).

The painful form is caused by spasm of the pylorus, increased peristalsis of the stomach, elevated gastric secretion in spasm of the cardial sphincter, owing to which no belching is noted. Pains and dyspepsia disappear upon the opening of the cardial sphincter and pylorus. Sometimes pains may result from perigastritis and periduodenitis. In these cases pain grows worse in walking, jolty riding, and straining because of irritation of the peritoneum. Pains may also be the result of a sharp reduction in the sugar content of the blood (hypoglycaemic reaction). In such cases pains are accompanied by severe general debility, facial pallor, tremor of the hands, a feeling of hunger, and cold sweats.

The painful form is differentiated from gastric ulcer and gastritis with secretory insufficiency. In the latter case, pains are felt shortly after a meal and in the painful form, with an increased or retained secretion, 2-3 hours after a meal.

In gastritis with a normal or increased secretion, constipation is a frequent feature, apparently as a result of overstimulation of the vagus nerve. Simetimes gastritis is concurrent with a disease of the bile ducts and liver, in which case it is of a secondary reflex character.

The patient's condition is quite satisfactory. Only in case of a prolonged lenient diet may signs of vitamin C deficiency make their appearance, such as dry skin, succulent gums, etc. There is also a nervous derangement, especially in the vegetative system, which is manifested by coldness of the extremities, acrocyanosis, proneness to sweats, etc.

Gastric juice with normal or increased acidity. In the gastric contents after Boas-Ewald's test breakfast, the ratio of solid parts to the liquid ones is 1 : 5. The solid part is adequately fragmented, while the liquid part is turbid and emits an acid odour. Bile and blood as an admixture are met with only occasionally. The urine is sometimes found to contain a large amount of phosphorus, possibly due to vegetative disturbances. The faeces contain little undigested matter. As distinct from gastric and duodenal ulcer no occult blood is found.

X-ray examination reveals wide, rigid folds, which tend to lose their regular pattern. The mucous membrane has a rough granular relief. These changes are especially frequently encountered in the pylorus and the duodenum (gastroduodenitis or pyloroduodenitis). In gastritis complicated by an adhesive process a deformity is found at the exit of the stomach or in the duodenum.

During therapy food must be bland to avoid mechanical and chemical damage. It is forbidden to eat spicy and fatty foods, bread, sauerkraut, sour milk, sour fruits, much meat and spices to avoid an increase in hydrochloric acid secretion. It is indispensable to abstain from tobacco and alcohol. The diet prescribed consists of milk gruel of semolina and rice, vegetable purée, white (wheat) bread, rusks, milk, cottage cheese, butter, boiled meat, chicken, and fish. A good effect is produced by mineral water which is drunk by mouthfuls an hour before a meal. Alkalis are prescribed, such as magnesia or soda, to reduce acidity.

GASTRIC AND DUODENAL ULCER (ULCUS VENTRICULI ET DUODENI)

Ulcer of the stomach and duodenum is a fairly frequent occurrence. It runs a chronic course and is responsible for a considerable loss of the working capacity and even for complete disability. In the past, this disease was considered to be a local lesion, but studies carried out by Russian authors (M. Konchalovsky and others) made it possible to establish that a gastric or a duodenal ulcer is a systemic disease affecting the entire body.

Aetiology and pathogenesis. Many theories have been advanced to explain the aetiology of ulcer. Virchow asserted that gastric ulcer is consequent upon thrombosis of the gastric arteries and erosion of the necrotized area by hydrochloric acid. Aschoff believed that predominant localization of an ulcer to the small curvature is associated with a mechanical and chemical damage to the gastric mucosa by food entering the stomach along the small curvature. Advocates of the peptic theory attributed the genesis of ulcer to an increased digestive capacity of gastric juice. Some authors attribute ulcer to injury sustained by the mucous membrane due to spasm of smaller vessels under the influence of reflex stimuli from the pathologically changed gallbladder, vermiform appendage, genitalia, etc. Finally, for a long time a gastritic theory was current, according to which ulcer was secondary to antecedent inflammatory changes in the gastric mucosa, which often cause superficial ulceration developing under appropriate conditions into a round ulcer.

Actually, hyperacidity is indispensable for ulcer formation. Undoubtedly, mechanical and chemical factors play a role, since ulcer often affects persons with unhealthful dietary habits, indulging in drinking and smoking. The fact is also known that gastritis and diseases of the liver, bile ducts and vermiform appendage are often precedent to ulcer. Nevertheless, clinical practice has shown that the above-mentioned factors do not necessarily entail ulcer formation. The authors of the theories indicated above disregarded the significance of the nervous system in the genesis of ulcer. The Soviet scientists N. Burdenko, L. Koreisha, A. Speransky, and B. Mogilnitsky proved the existence of an association between a lesion of the central and peripheral nervous system and the development of ulcer. The neurogenous theory of the pathogenesis of ulcer was developed further into the corticovisceral theory by K. Bykov and I. Kurtsin who proved that gastric and duodenal ulcers may result from disturbances in the central nervous system, i.e., the brain cortex.

According to this theory, the brain cortex under the influence of external and internal stimuli sends off impulses to the stomach and the duodenum, which cause a spastic contraction of vessels, resulting in a local trophic disorder followed by erosion of the affected area by the gastric juice.

In the majority of cases ulcer is encountered in persons who are easily excitable and very irritable. It often develops after an intense emotional strain (fear, distressing anxiety, etc.).

One should not, however, underestimate the importance of an irregular diet, which in combination with an emotional strain contributes to the onset of ulcer.

Pathologic anatomy. Gastric ulcer is most commony located in the pyloric part and along the small curvature, and sometimes in the cardial part. It has sharp edges and assumes the shape of a funnel, its apex being directed into the depth of the stomach wall. An ulcer is round or oval, and is usually isolated, while multiple ulcers are encountered very rarely.

The floor of an ulcer is covered with fibrin, decomposed cells and mucus. In case of inflammatory changes in the serous coat of the stomach, caused by the formation of an ulcer, adhesions to the adjacent organs develop—to the gallbladder (pericholecystitis), the duodenum (periduodenitis), etc. Adhesion of the stomach to the adjacent organs changes its form; sometimes deep scars and retractions form a bilocular stomach.

In respect of its anatomic structure, an ulcer may be simple (ulcus simplex), round (ulcus rotundum), penetrating (ulcus penetrans), when an ulcer penetrates into the region of the liver or the omentum, and perforating (ulcus perforans), when it perforates into the free abdominal cavity. Sometimes extensive thickenings are formed around an ulcer, its edges becoming hard and callous; such ulcers are known as callous and are attended with intense, constant pains.

Clinical picture. Gastric and duodenal ulcers are more frequent in men than in women, mainly at ages of 25 to 40 years. A carefully conducted interview with the patient is highly important for the diagnosis. This disease is characterized by pains, haemorrhages, vomiting and dyspeptic symptoms: heartburn, belching, etc. Pain is the principal symptom of ulcer. In uncomplicated cases it is located in the epigastrium, in duodenal ulcer, somewhat to the right of and upwards from the navel, at the edge of the rectus abdominis muscle. Pains are usually dull in character, sometimes gnawing, burning, and at the onset of the disease grow worse only after ingestion of coarse food. Later, after eating any, even bland, food they set in within 1-2 hours of a meal. Late, overnight, or so-called hunger pains are felt in duodenal ulcer as well as in gastric ulcer located near the pylorus. The origination of pains is attributed to irritation of the gastric mucosa by the acid gastric juice and spasm of the pylorus; therefore, aggravation of pains is noted at the height of the digestive process. Late pains are ascribed to irritation of the gastric mucosa after evacuation of the gastric contents by a continuous intensified secretion of gastric juice in the neurohumoral phase. The gastric mucosa becomes particularly sensitive when it is inflamed. Pains are relieved after ingestion of such foods as require a large amount of gastric juice, for example, milk, rusks, etc. That the appearance of pains is facilitated by the presence of hydrochloric acid is also confirmed by their considerable abatement after vomiting. That is why many patients feeling intense pains induce vomiting by artificial means in order to relieve their distress. Pains substantially change in character in cases where the disease process extends to the peritoneum, and signs of irritation of the peritoneum, muscular defence come on the stage. Continuous pains radiating to the back are felt in penetrating ulcers. Pains due to ulcer occur periodically and are intermittent in occurrence.

Vomiting is not an inevitable feature of ulcer. Usually, it occurs at the height of digestive activity and is encountered with complicated ulcers producing extensive adhesions to the adjacent organs, for example, the gallbladder (in stenosis of the pylorus). Vomitus contains remnants of food, sometimes bright-red blood, or has the colour of coffee dregs.

The appetite in an ulcer patient is usually retained, and it is only fear of intense pains appearing after a meal that makes him abstain from food. In some cases this may result in a loss of weight to the degree of emaciation.

The acidity and secretion of gastric juice in ulcer patients are as a rule elevated. Total acidity rises to 90 and higher, while free hydrochloric acid is equal to 70 instead of the normal 60-40. Examination of the gastric juice collected with a thin probe may reveal a considerable hyperacidity by the end of the second hour of the procedure.

Ulcer sometimes runs its course with a normal and even decreased acidity. A high-degree hyperacidity is usually a feature of duodenal ulcer. Hyperacidity of the gastric juice may be responsible not only for severe pains but also for heartburn and acid belching. An ulcer patient has a copious salivation and an acid taste in the mouth. The blood count is normal or shows erythrocytosis and polyglobulia, especially in duodenal ulcer.

The most frequent disturbance in the intestinal tract is constipation caused by vegetative derangements as well as a lenient diet. The patient's faeces are periodically found to contain occult blood. In view of the fact that occult blood may be found in the faeces of healthy persons eating meat and fresh greens the patient should be maintained on a milk and vegetable diet during three days prior to examination.

The motor activity of the stomach is as a rule unimpaired in ulcer, but in certain cases it is reduced, apparently due to spasm of the pylorus.

An ulcer gives direct and indirect roentgenologic symptoms (Figs. 56 and 57). Its principal direct sign is a niche—bulging of the stomach contours. In the presence of a defect in the mucous membrane the contrast substance is retained in the depression and is seen as a dark outward projection of the stomach contours of variable form. Sometimes air finds its way into the niche. The niche is usually located on the anterior or posterior surface of the small curvature. In duodenal ulcer the niche is encountered in the bulb and in the upper horizontal portion of the duodenum. Among the indirect signs is the so-called index finger symptom, i.e., a deep groove

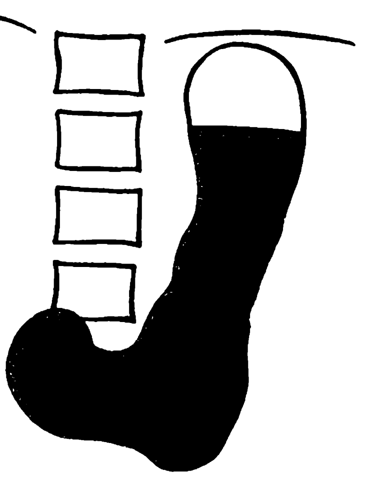
Fig. 56. X-ray of a normal stomach

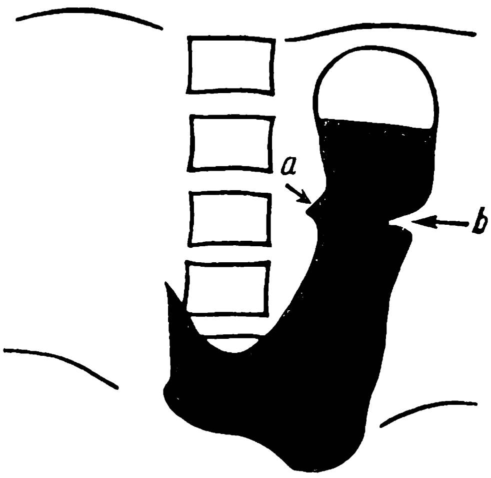

Fig. 57. X-ray of gastric ulcer
a—niche symptom; *b*—index finger symptom

in the contour due to a spastic contraction of the muscular lining (Fig. 57), found on the side opposite to the site of the ulcer. This retraction sometimes divides the stomach into two halves (bilocular or hour-glass stomach). The duodenum also suffers deformation in case of spasms and adhesions ("butterfly", "trefoil" symptom).

The course of ulcer varies with age and sex, location of an ulcer and the patient's individual characteristics. At a juvenile age, an ulcer runs its course without striking clinical manifestations. In old persons fresh ulcers are encountered rarely but they are often complicated by considerable haemorrhage resulting from sclerotic changes in the stomach. Combination of ulcer with gastritis and cholecystitis substantially changes the clinical picture of the disease.

Ulcer runs a chronic, cyclic course characterized by relapses and remissions. Therapy results in recovery in 3-4 weeks, the niche healing within 4-6 weeks. A remission lasts from 6 to 12 months and longer; there are cases, however, of a drastic shortening of intervals between relapses. Exacerbation of an ulcer, particularly that of the duodenum, is commonly observed in spring or autumn. In the periods of remission the patient is fully capable of working.

Complications of ulcer. In certain patients ulcer is accompanied by severe complications: haemorrhage, perforation, stenosis of the pylorus, malignization, etc.

A rupture of blood vessels at the site of an ulcer and subsequent bleeding is associated with physical or nervous overstrain, as well as with a digression from the dietary regimen, and is attended with acute anaemia marked by dizziness, dimness of vision, nausea and dyspnoea. The patient grows pale, his extremities become cold to the touch, the pulse becomes rapid, and the arterial pressure falls. Vomiting of blood (haematemesis) or the discharge of a pitch-like stool (melaena) confirms the presence of haemorrhage. Bleeding is followed by leucocytosis and fever, but hyperaemia and oedema at the site of the ulcer diminish, owing to which pains subside considerably. Death from bleeding is quite infrequent. Prognosis becomes ominous in a progressive decline of the arterial pressure and growing anaemia. It is necessary to consider the presence of other diseases likely to cause copious bleeding: gastric cancer, liver cirrhosis, etc.

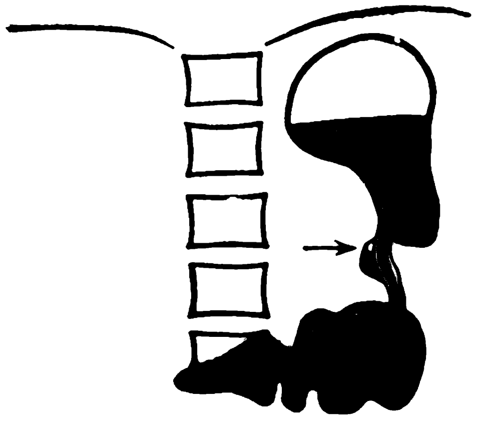

Fig. 58. Hour-glass stomach. A niche indicated by arrow is seen on a small curvature

Perforation of an ulcer is a formidable complication and is mainly encountered in men. It is twice as frequent in duodenal ulcer as in gastric ulcer. The process of perforation consists in penetration of an ulcer to the serous coat of the stomach or duodenum and its rupture into the abdominal cavity. The onset is sudden, with an excruciating pain like that felt by one stabbed with a dagger, and a picture of collapse: cold sweats, small pulse, pallor, a sharp fall of the arterial pressure, and shallow breathing. These symptoms are soon joined by clinical signs of diffuse perforative peritonitis. The abdominal muscles become rigid and take no part in respiratory movements. There is intestinal paresis, no sounds are heard on auscultation of the abdomen, and the hepatic dullness disappears, since gas fills the entire abdominal cavity and displaces the liver upwards. Fever is present. Sometimes the perforated part is covered by the omentum to form a covered perforation likely to become incapsulated and suppurate. The patient can be saved by emergency surgery; the mortality rate increases with every hour of delay in operation.

Stenosis of the pylorus may be functional and organic in character. Functional strictures result from periodic spasms; organic strictures, from deep cicatricial changes. Sometimes these scars divide the stomach into two cavities (so-called hour-glass stomach; Fig. 58). With an organic cicatricial stenosis of the pylorus, the patient has a sensation of fullness and heaviness in the epigastrium. Examination of the stomach region may reveal peristaltic and antiperistaltic waves, the patient complaining of vomiting, acid belching, a foul taste in the mouth, growing debility, oliguria, and constipation. Vomitus contains remnants of earlier ingested food. As the stomach muscles weaken gastroptosis and distention of the stomach develop. There is extreme emaciation, the skin is dry because of dehydration, and the tongue is dry and coated. The stomach bulges and its boundaries can be determined visually. On an empty stomach and sometimes 5-6 hours after a meal, palpation may elicit a splashing sound. X-rays reveal retention of barium during 24 hours and longer.

A lack of common salt and calcium in the blood and tissues produces hyperexcitability of the neuromuscular apparatus, derangements in the central nervous system with painful spasms of the skeletal muscles, and finger cramps in the form of an obstetrician's hand (gastric tetany). An organic stenosis may sometimes be mistaken for a functional spasms of the pylorus due to inflammation. In such cases, atropinization helps eliminate the functional construction.

Prophylaxis of cicatricial stenosis in ulcer consists in an early treatment by means of surgical establishment of a gastrointestinal anastomosis. Large callous ulcers of the stomach undergo malignant degeneration in 10 per cent of cases. Malignization of a duodenal ulcer is extremely rare. Signs of malignization are a progressive decline of the acidity of gastric juice, weight loss, anaemia, and change in the character of pains which become permanent. The appetite is reduced; occult blood is always present in the faeces, as distinct from ulcer.

Prognosis and work capacity. The patient becomes disabled in periods of exacerbation. During a remission he is able to perform his regular job in the absence of physical and emotional overstrain and with observance of dietary rules. In case of complications or frequently relapsing forms of ulcer, the patient should be considered incapacitated or forbidden to perform work involving physical and mental overstrain as well as night jobs.

Treatment and prophylaxis. Ulcer is prevented by measures to forestall nervous shock, by abstinence from coarse food and adherence to a regular diet, by rational organization of recreation and provision of hygienic conditions. A role of great importance in ulcer prophylaxis is played by timely treatment of all functional (neurosis) and organic (gastritis) diseases of the stomach, which may contribute to the genesis of an ulcerative process.

A course of treatment lasts from 4 to 6 weeks. During therapy the patient should have complete bed rest; he is safeguarded against anxiety by institution of a protective regimen. The diet should contribute to a reduction in the secretion of gastric juice and abatement of the inflammatory symptoms accompanying ulcer. For this purpose, pickles, extractive foods, rich soups, broth, smoked foods, tinned foods, marinades, etc., should be eliminated from the diet. Food must be bland to avoid the risks of mechanical and chemical injury; it should be eaten at regular intervals in moderate quantities and not be very cold or hot. There are a number of schedules of dietotherapy recommended for uncomplicated ulcer, which are mainly based on the above-mentioned principles. A. Yarotsky proposed a protein-butter diet consisting of egg whites and butter. He recommends to begin treatment with one egg white and 20 g of butter daily, adding on each subsequent day one egg white and 20 g of butter so that on the 10th day of treatment the patient would receive 10 egg whites and 200 g of butter. But for all its positive qualities, Yarotsky's diet is poorly tolerated by patients. It is better to conduct dietotherapy according to the schedule used at Soviet medical institutions and known as diets No. 1*a*, No. 1*b*, and No. 1. Under this schedule the patient receives about 2,000 Cal during the first 2-3 weeks (diet No. *1a*) in six meals a day. The diet consists of milk, milk cream, sugar, soft-boiled eggs, mucilaginous milk soups cooked of cereals, sweet kissels, jelly, a light cake of wheat flour, nonsour fruit and berry juices, and unsalted vegetable juices. In the next two weeks, diet No. *1b* is given which includes foods from diet No. *1a* with an addition of liquid milk gruels (rice, semolina, oatmeal, etc.), puréed meat and fish (quenelles, souffle) with an addition of milk and butter, white rusks in quantities of 50 to 200 g daily, weak sweet tea, and cocoa. From the 4th week, the patient is transferred to diet No. 1 which additionally includes vegetable soups, vegetable purée, thin meat broths, lean boiled meat and fish served as steam-cooked cutlets or in one piece, cottage cheese, sour cream, compotes, fresh white bread, and common salt in daily quantities of up to 5 mg. Diet No. 1 is recommended for 3-4 months. The food of an ulcer patient should in all stages of the disease contain vitamins, mainly vitamins B and C. Tobacco and alcohol are forbidden.

To neutralize hyperacidity, patients willingly take soda; later, however, when absorbed into the blood, it stimulates an increased stomach secretion. A better effect is produced by the administration of magnesium oxide and magnesium carbonate in a dose of 1-2 g 3-4 times daily, sometimes in combination with belladonna half-an-hour before meals (formulas Nos. 78, 79). To intensify mucus secretion and diminish the action of acid on the stomach wall, bismuth is prescribed in a dose of 3-4 g to be taken in 24 hours. Administration of atropine and its preparations (formula No. 90) reduces the juice secreting effect of the vagus nerve and eliminates spasms caused by its stimulation. To reduce hyperexcitability of the brain cortex and subcortical centres and suppress pathologic impulses transmitted to and from the stomach, luminal in a dose of 0.05 g to be taken 1-3 times daily, a 10 per cent sodium bromide solution to be introduced intravenously in a dose of 10 ml once daily during a month, a 0.5 per cent novocain solution (10 ml, 15-20 injections, and ganglioblocking agents: ganglerone, hexonium, etc., are recommended. The new preparations roter and vicaline have been used during the past few years.

For the same purpose, treatment by sleep, as well as vitamin B_1 therapy (formula No. 156) have been employed in recent years. Vitamin B_1 is administered subcutaneously in a daily dose of one ampule for a month.

Drug therapy is combined with physio- and balneotherapy, which is not allowed in case of haemorrhages. Pains are allayed by a local application of heat in the form of hot-water bottles or heaters, poultices, etc. Quartz light in an erythema dose, diathermy, pine baths, applications of paraffin, ozocerite, etc., are employed.

To speed up the healing of an ulcer stimulation treatment is used. Pasteurized milk, blood of a heterologous group in doses of

3-5-10 ml every 2-3 days, cod liver oil, and aloe are injected intramuscularly.

Treatment at a sanatorium and health resort produces a beneficial effect on the nervous system which is favourably influenced by various factors: rest, salubrious climate, well-regulated therapeutic diet, mineral water, and entertainments. Health resort theapy is contraindicated for stenosing, complicated and fresh ulcers, especially those attended with haemorrhages.

Indications for surgery may be relative and absolute. Among the former are stubborn and prolonged pains reducing work capacity, a progressive decline in acidity and weight, callous ulcers liable to malignization, absence of response to a prolonged dietotherapy and regimen, etc. The absolute indications for an operation are perforation of an ulcer, a prolonged copious gastric haemorrhage, a far-advanced stenosis of the pylorus, and malignant degeneration of an ulcer.

The remedies for gastric or intestinal haemorrhage are strict confinement to bed, a fasting diet for 3-4 days, application of ice to the abdomen, and parenteral administration of haemostatic drugs: horse serum in a dose of 15-20 U or a 10 per cent calcium chloride solution. In case of repeated haemorrhage, blood transfusion is helpful while surgery should be employed only when the abovementioned measures have proved a failure.

During haemorrhage, it is recommended to offset dehydration of the body by drip enemas of physiologic salt solution and intravenous and subcutaneous injections of a glucose solution. When bleeding has ceased, the patient is given cold food: milk cream by teaspoonfuls, butter in pieces, and cool milk.

CARCINOMA OF THE STOMACH (CANCER VENTRICULI)

Gastric carcinoma is a frequent form of cancer responsible for approximately 35-40 per cent of all deaths from malignant tumours.

Carcinoma of the stomach is more common in men than in women, and mainly affects elderly and old persons, although it may also be encountered at a young age. The highest incidence is noted at ages of 50 to 60.

Aetiology and pathogenesis. The aetiology of cancer is unknown. Highly important contributing factors in the genesis of carcinoma are several antecedent pathologic conditions, the so-called precancerous diseases, such as benign tumours, ulcer of the stomach, particularly the callous form, gastritis, and stomach polyps. The latter, according to evidence supplied by different authors, undergo malignant degeneration in 12-60 per cent of cases; leiomyoma, lipoma, and fibromyoma rarely develop into a carcinoma.

The question of chronic gastritis being a precursor of cancer has long been a subject for debate. Some authors regard gastritis as a precancerous state, others, as a parallel or concomitant disease process. At the same time, clinical evidence confirms the frequency of gastric carcinoma in persons with a history of chronic gastritis. It is beyond doubt that changes in the stomach mucosa (sometimes with reconstruction of the glandular apparatus, as is the case in atrophic gastritis) may become responsible for the genesis of cancer.

According to certain data, a role in the development of stomach carcinoma is played by abuse of too hot or too cold foods, cold aerated drinks, smoking and alcohol.

In the past few years, a virus theory of cancer, gastric carcinoma in particular, has been proposed. This theory, however, although supported by many, has quite a few opponents.

The importance of hereditary factors in the genesis of cancer cannot be regarded as proved.

Pathologic anatomy. Gastric carcinoma is most commonly located in the pyloric portion, less often in the small curvature, in the cardial part, and rarer still on the large curvature. One should differentiate between polypous, primary ulcerative, mucous (colloid) carcinoma, and scirrhus (a hard, fibrous carcinoma). In respect of its histologic structure, carcinoma of the stomach is subdivided into glandular or cylindrical (adenocarcinoma), and acinous or medullary, carcinoma. Polypous cancer develops from gastric polyps (from the mucous membrane) in the form of a node attached by a pedicle and resembling a cauliflower. Sometimes, a polypous carcinoma is flattened like a mushroom (fungous carcinoma). In ulceration of a fungous or medullary carcinoma, the edges round the ulcer are raised and the tumour becomes saucer-shaped or cup-shaped. A mucous carcinoma is a jelly-like mass which infiltrates the wall of the stomach and its muscular lining. A fibrous carcinoma (scirrhus) involves a considerable development of interstitial connective tissue which lends the tumour a hard consistency. A scirrhus infiltrates the stomach wall, the stomach becomes narrowed, contracted, and smaller in volume while the pylorus is converted into a dense tube. Decomposition of a malignant tumour and its ulceration give rise to bleeding and purulent and putrefactive processes, sometimes with perforation of the stomach followed by purulent peritonitis. Perforation of the anterior abdominal wall produces an external fistula, and perforation into the intestine, a gastrointestinal fistula.

Metastases. Cancer cells or tumour particles break off and are carried with the lymph and blood to the mesenteric lymph nodes, omentum, peritoneum, intestine, as well as to the distal organs: the lungs, the kidneys, the muscles, the bone system, the brain, etc. Gastric carcinoma is prone to produce metastases into the left supraclavicular lymph nodes located near the heads of the sternocleidomastoid muscle (Virchow's gland) and into the left axillary lymph nodes. Sometimes a gastric carcinoma encroaches upon the liver, the pancreas, the abdominal portion of the oesophagus, and the portal fissure (porta hepatis). Through the lymphatic routes gastric cancer invades the peritoneum producing tiny nodules (miliary carcinoma). Metastases are also found in the skin near the umbilical orifice and in the ovaries (Krukenberg's tumour). Extension of cancer to the peritoneum usually entails haemorrhagic or serous ascites.

Clinical picture. Cases of asymptomatic carcinoma are quite frequent. The tumour is discovered by chance (8-10 per cent of cases) in persons who have died from other causes. At the same time, a number of findings (inexplicable loss of appetite and weight, a feeling of discomfort after ingestion of food, fullness and pressure in the epigastrium after a meal, growing debility, and reduced vitality) should alert the physician to the possibility of a malignant neoplasm.

The clinical symptoms of gastric carcinoma vary with the stage of its development, localization and spread through the lymph nodes and visceral organs. The principal symptoms of gastric carcinoma are as follows: a disturbance in gastric digestion, epigastric pains, growing anaemia and loss of flesh, sometimes vomiting of blood.

The appetite is reduced or absent altogether. Aversion to meat and protein foods is noted frequently. Sometimes, particularly in case of a tumour of the pylorus, the patient vomits mucus and undigested food immediately after a meal. In the presence of bleeding vomitus may contain bright-red blood or have the colour of coffee dregs due to conversion of haemoglobin into methaemoglobin. Sometimes vomited matter emits a foetid odour from a decomposing tumour.

Pains are nagging and are felt in the epigastrium, a distressing sensation of pressure in the stomach region being also present sometimes. Pains may become excruciating when a tumour invades the pancreas, in peritonitis, and metastases into the bones.

On inspection, one notes a yellowish-grey colour of the face, a loss of lustre and an apathetic look in the eyes, and emaciation to the degree of cachexia. It will be recalled that in symptomatic therapy there may be no loss of flesh, and sometimes even a gain in weight is notable. Cachexia is the most rapid to appear when the tumour is localized in the cardia or the pylorus. In certain cases peripheral oedema develops due to malnutrition and haemorrhages which is associated with hypoproteinaemia (hypooncotic oedema). A large tumour, as well as its metastases into the liver, is sometimes detectable by a visible distension of the epigastrium and right hypochondrium. With a carcinoma located in the pylorus, intensified peristaltic and antiperistaltic movements of the stomach are visible. External examination enables to detect ascites. In certain cases it is possible to palpate solid metastases into the cervical, axillary and subclavian lymph nodes (Virchow's gland), as well as metastases into the skin on the navel. It is best to palpate the stomach with the patient first standing and then lying down and after it has been emptied by lavage or administration of a laxative, especially when the tumour is located in its posterior wall. Tumours are more easily palpated when the abdominal wall is soft; in such cases a dense knobby formation, painless and displaceable in respiration (if the tumour is not adherent to the anterior wall of the abdomen) may be revealed. Sometimes, metastases to the omentum and the lymph nodes rather than the stomach tumour itself are palpable in the stomach region. In case of metastases into the liver, an enlargement is noted mainly in its left lobe.

The gastric juice. The gastric juice contains much mucus, leucocytes, desquamated epithelial cells of the gastric mucosa, and tumour cells. In 75 per cent of cases gastric secretion is absent or diminished, in 40 per cent histamine refractive achylia is present due to atrophic changes in the gastric mucosa. A characteristic feature of cancerous gastric achylia is a wide range of discrepancy between total acidity and the level of free hydrochloric acid. This is accounted for by the presence of a considerable amount of organic acids as well as free hydrochloric acid being partly neutralized by food proteins, effused blood, and the tissue of the tumour itself. It is highly important to detect lactic acid in the gastric contents since it is found only in the absence of free hydrochloric acid, with a disturbance in the evacuative function of the stomach, and in the presence of lactic acid fermentation. Lactic acid may also be present in case of stenosis of the pylorus due to various causes, including benign tumours, when the stomach evacuation is delayed.

Microscopy of the gastric contents reveals Boas-Oppler lactic acid fermentation bacilli. Occult blood is detected in the faeces by means of a benzidine test in 90 per cent of gastric cancer patients. It is important diagnostically when it is present constantly and is even observed when meat, fish and greens are excluded from the diet, and when there is no danger of damage with a tube. Anaemia rarely occurs in the early period of the disease, and develops later as a result of a blood loss, inadequate assimilation of food and iron, and general intoxication. Anaemia may be hypochromic and iron deficiency, with poikilocytosis, anisocytosis, neutrophilic leucocytosis and an elevated ESR. Metastases into the bone marrow give rise to hyperchromic pernicious anaemia. At the same time, a burning sensation in the tongue and obliteration of its papillae (Hunter's glossitis) are encountered.

A rise in body temperature takes place when the disease is joined by an inflammatory process in the stomach, decomposition of the tumour, and resolution of foreign protein.

X-ray examination is extremely important for the diagnosis of gastric carcinoma. X-rays reveal the so-called filling defect (Fig. 59). In carcinomatous infiltration peristalsis is absent. In infiltration

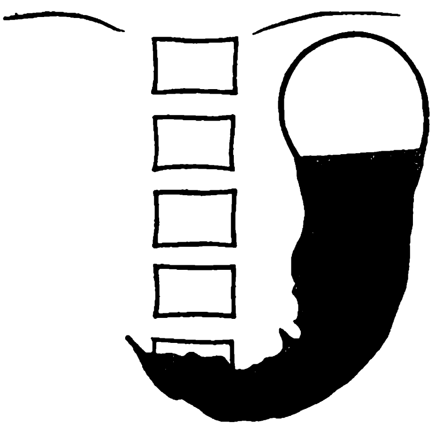

Fig. 59. X-ray of gastric carcinoma

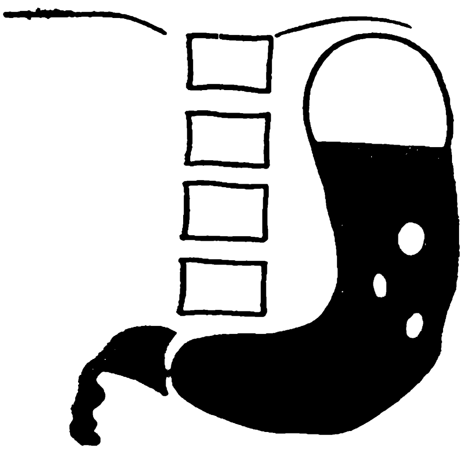

Fig. 60. X-ray of gastric polyposis

of circumscribed areas dead zones are discovered, i.e., areas of absent stomach peristalsis. In case of a scirrhus the stomach assumes the shape of a tube. In polyposis (Fig. 60) large filling defects are visible. A stomach adherent to the adjacent organs is not displaced either spontaneously or by palpation.

Depending on the predominance of a particular syndrome the following clinical forms of gastric carcinoma are distinguished: febrile, anaemic, painful, painless, haemorrhagic, latent, and oedematous. A knowledge of these forms is helpful to diagnosis. Sometimes, a subfebrile temperature or a high-grade fever is the first symptom of the disease. The painful forms of cancer of the stomach are attributed to primary ulceration. In certain cases, an existing tumour is disclosed for the first time by a profuse gastric haemorrhage. A prolonged, usually external, bleeding leads to severe anaemia.

Course. At the onset gastric carcinoma runs a latent course and has but little effect on the patient's general condition. An aggravation becomes apparent with the development of metastases or various complications. The duration of the disease depends on the localization and size of the tumour. A carcinoma located at the entrance to or exit from the stomach (in one of every five cases) early leads to severe functional disturbances, emaciation and death. The cause of death may be a massive gastric haemorrhage, adhesion of the stomach to the large intestine and formation of a fistula between them, with an exhaustive diarrhoea, or perforation of the tumour into the free abdominal cavity with the development of peritonitis (infrequently). More commonly there is perforation into the pancreas, adhesion of the stomach to the adjacent organs (liver, intestine, etc.). In young persons cancer runs a more malignant course than in old age. The duration of gastric cancer from the appearance of its first manifestations to death is not longer than 1-2 years.

Differential diagnosis. It is very difficult to identify gastric carcinoma in the initial period of the disease, whereas in the final stage the diagnosis is easier to make because the tumour is palpable and its cardinal manifestations are obvious: anaemia, cachexia, and x-ray symptoms.

An early diagnosis is made possible by health education, prophylactic treatment of elderly and old persons suffering from achlorhydria, partial or total loss of appetite, loss of weight, and digestive disorders. During the past few years, gastrobiopsy by means of a probe with a special contrivance for cutting out pieces of gastric mucosa for subsequent histologic examination has been employed. For the same purpose, lavage of the stomach (after M. Yasinovsky) is carried out or special bags which, when filled with air, cause exfoliation of mucosal cells are inserted into the stomach together with a probe. These cells are subjected to a histologic study (exfoliative cytology).

Roentgenologic and gastroscopic examination, constant presence of occult blood in the faeces, and examination of the gastric juice furnish the required evidence for an early identification of gastric carcinoma.

Gastric carcinoma should be differentiated from gastritis and stomach ulcer. Evidence in favour of uncomplicated forms of stomach ulcer are an undulating course, periodic pains, an increase of the secretory and motor function of the stomach, and a niche discovered on x-ray examination. Chronic gastritis is distinguished by a protracted course. Cancer should be considered in gastritis when there is a progressive loss of appetite and developing anaemia. On palpation of a visible tumour in the stomach region, gastric carcinoma is most commonly confused with primary cancer of the liver, the bile ducts, or the intestine. Sometimes, in the presence of a high-grade fever accompanying a decomposing gastric carcinoma, a wrong diagnosis of pulmonary tuberculosis or malaria is made. An erroneous diagnosis of cancer may be made in diseases running their course with general emaciation: alimentary dystrophy, vitamin deficiency, hypophyseal cachexia, etc. Sometimes, tumours of the abdominal wall and the abdominal cavity—lipomas, a displaced kidney, and enlarged lymph nodes, are mistaken for carcinoma. It is sometimes extremely difficult to differentiate between gastric carcinoma and sarcoma, gumma, or tuberculosis of the stomach.

Prognosis depends on the stage of gastric cancer. In the first stage, there is a small circumscribed neoplasm in the depth of the mucous membrane and submucous lining without regional metastases. In the second stage, the tumour grows into the muscular layer but fails to encroach upon the serous membrane and is not adherent to the adjacent organs. In the third stage, the tumour grows to a considerable size, invades the adjacent organs, while its mobility is markedly limited and numerous metastases to the regional lymphatic glands are present. In the fourth stage, the tumour metastasizes to distal organs. Prognosis may be favourable in the first and second stages of the disease when a radical surgical intervention may yield quite satisfactory results.

Prophylaxis. Among the prophylactic measures great importance is attached to a thorough therapy of long-standing ulcers, especially callous ones, which are liable to malignant degeneration, a systematic therapy of chronic gastritis, regular observation of patients with achylia, and surgical treatment of polyps.

Treatment. The diet should be adequately nourishing, rich in vitamins and correspond to the secretory function of the stomach. Bitters are prescribed to whet the appetite (formulas Nos. 73, 74 and 74*a*). Food must be eaten in fractional quantities, in liquid or half-liquid form (milk, cocoa, chocolate, kefir, minced meat, brains, fish, vegetable purée, and bland farinaceous and sweet foods). Systematic lavage of the stomach is recommended to prevent vomiting and improve the appetite. Hydrochloric acid, pepsin (formula No. 74), and pancreatin (formula No. 75) are also administered. Other remedies are: repeated blood transfusion, narcotics to relieve pains (at first, luminal and codeine, later, pantopon and morphine), and in anaemia, raw liver and vitamin B_{12}.

DISEASES OF THE INTESTINE

An Outline of Anatomy and Physiology

The intestine extends from the pyloric opening of the stomach to the anus. It consists of the duodenum, the small and the large intestine.

The duodenum is attached to the posterior wall of the abdominal cavity, and is shaped as a horseshoe. Its upper portion lies horizontally, and is an extension of the pylorus. Next is the vertical portion, which is continuous with the lower horizontal portion communicating with the small intestine. The vertical portion of the duodenum receives two ducts: the pancreatic duct and the common bile duct. Both ducts open out into Vater's ampulla. The small intestine measures 7-8 metres in length. The upper two thirds of the small intestine are known as the jejunum, the remaining portion of the small intestine is the ileum. The ileum forms a number of loops. It is attached to the posterior wall of the abdominal cavity with the mesentery. The small intestine is separated from the large intestine by the valve of Bauhin.

The wall of the small intestine consists of three coats: the outer, the middle, and the inner. The outer, serous, coat is formed by the peritoneum extending from the mesentery by which the intestine is suspended. The mesentery is short in the initial portion of the small intestine and is lengthened at the end of the ileum. The middle coat consists of two muscular layers arranged in two directions: lengthwise in the outer layer, and circular in the inner layer. The inner, or mucous, coat forms a large number of circular folds covered with villi which have an extensive capillary and lymphatic network. Between these villi are lodged Brunner's and Lieberkuehn's glands secreting the enzyme erepsin. The mucous membrane also contains lymphatic glands—solitary follicles, which in the lower part of the small intestine form aggregations—Peyer's patches consisting of groups of Brunner's and Lieberkuehn's glands. The mucous membrane with its villi is covered by a simple columnar epithelium consisting of cells which absorb food and goblet cells which secrete mucus. The large intestine also has three coats: the mucous, the muscular, and the serous. The mucous coat has no villi; its surface, however, is also lined with simple columnar epithelium consisting of goblet cells. The large intestine, which is 1.5 metres in length, takes its origin from the small intestine in the right iliac region. It is subdivided into the caecum into which the vermiform appendage opens out, the ascending portion (colon ascendens), the transverse portion (colon transversum), the descending portion (colon descendens), the sigmoid colon (sigma), and the rectum. The terminal part of the intestine forms the anus, which is provided with two sphincters: the external and the internal. The ascending colon begins in the left iliac region, passes upwards to the liver and forms the so-called hepatic flexure (flexura coli hepatica s. dextra); an identical bend is found at the splenic region (flexura coli lienalis, s. sinistra). The transverse colon lies across the abdomen as a curved line or a sagging garland. The ascending and descending portions of the large intestine are covered with the peritoneum only at the front, while the other parts are covered on all sides.

The intestine is innervated by the vagus and sympathetic nerves, which form nerve plexuses: the solar plexus (plexus solaris) and the mesenteric plexus (plexus mesenterialis). The intestinal submucous layer lodges Meissner's plexus, and the muscular layer, Auerbach's plexus. The intestine performs a number of functions: (a) digestion of food, begun in the stomach, is continued and completed in the intestine; (b) assimilation of digested food; (c) excretion and evacuation of indigested remnants of food.

Food is digested by the enzymes of the pancreas trypsin, lipase, and diastase, and by the enzyme of the small intestine, erepsin. Trypsin and erepsin break up proteins into amino acids which through the system of the portal vein are carried into the liver where they undergo further processing. Lipase splits fats with the aid of bile into glycerol and fatty acids, and diastase converts polysaccharides into soluble sugar which is taken up by the blood to form animal starch—glycogen. As distinct from the stomach, the intestine has an alkaline medium. Proteins, fats, and carbohydrates are converted to a soluble state under the action of enzymes and are absorbed in the small intestine.

The motor function of the intestine is performed by pendulous and peristaltic movements owing to which the intestinal contents are thoroughly mixed and come into close contact with the intestinal juice and the absorbing surface of the intestinal mucosa. By peristaltic movements, the intestinal contents are gradually pushed forward due to the contractions of the circular and longitudinal muscles of the intestine. Muscle contractions are stimulated by the vagus nerve and paralyzed by the sympathetic nerve.

The intestinal contents after being exposed to the action of enzymes are absorbed by the extensive surface of the small intestine, its villi. Proteins and carbohydrates (amino acids and monosaccharides) taken up by the villi enter the liver through the portal vein, while fats are absorbed by the lymphatic vessels and through the common thoracic duct (ductus thoracicus) are delivered into the systemic circulation.

In the ileum, caecum, ascending and transverse colon fermentation and decomposition processes take place owing to the bacterial flora which is present in a large variety here. The ileum is the site of fermentation of carbohydrates; the caecum and ascending colon, of protein decomposition processes. The motor function of the large intestine is identical with that of the small intestine. The secretory function of the large intestine is restricted only to the production of mucus. Nutritive substances are not absorbed within it at all, save for water and salt, which fact accounts for the consolidation of faecal matter within it. The large intestine also performs the function of excreting iron, mercury, lime, urea, etc. The act of defecation takes place in response to the stimuli conveyed from the rectus to the defecation centre in the lumbar part of the spinal cord. Faecal matter is evacuated by a simultaneous contraction of the rectal muscles and the abdominal muscles (prelum abdominale) and relaxation of the anal sphincters. All processes occurring in the intestine—secretory, motor and absorptive—occur under the direct influence of the central nervous system through its stimuli conveyed to the vagus and sympathetic nerves, the thoracoabdominal nerve, and the splanchnic nerves.

Impairment of the normal regulatory function of the central nervous system, nervous and mental shocks and overstrain very rapidly influence the function of the intestine in which serious organic changes ensue. The entire process of food digestion lasts 24 to 36 hours.

Cardinal Symptoms of Intestinal Diseases *

During an interview with the patient, it is necessary above all to clarify the succession of symptoms pointing to an intestinal disease, and a possible affection of individual organs, the stomach and liver in particular, and systems which may be probable causes

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* For examination of faeces see p. 84.

of the involvement of the intestine in the disease process. One must also disclose a past history of intestinal diseases and find out about the living conditions and personal habits, since an unhygienic manner of life is often responsible for serious diseases of this organ. Certain occupational health hazards also play a role in the genesis of intestinal disease, for example, constipation in persons leading a sedentary life, dyspepsia in persons living on dry foods, and lead colic in house-painters and printshop workers. A significant sign is a loss of weight which sometimes points to a malignant character of the disease. Special attention must be paid to the condition of the nervous system, emotional upset and nervous shocks. In addition to complaints of a diminished appetite, nausea and vomiting, it is also necessary to ascertain a number of important symptoms likely to be encountered in intestinal disease. Among them are diarrhoea, constipation, meteorism, pains along the intestine, and intestinal haemorrhages.

Pains. Pains are sometimes the principal symptom complained of. A thorough inquiry about the time of onset and the character of pains, their radiation, aggravation or abatement under the influence of a particular factor may be helpful to determine roughly the site, character and cause of the disease.

Pains are variable in character and origin. Paroxysmal pains (intestinal colic) occur periodically as a result of spasm of the smooth muscles. A colic may also be felt in the rectal region. It is manifested by rectal urgency, only mucus and blood, however, being discharged instead of faecal matter. This ineffective straining is known as tenesmus.

Pains due to an inflammation are persistent in character. They grow worse on straining when coughing or defecating, as well as when the disease process invades the serous membrane. In this case, pains aggravate on palpation. This occurs, for example, in perigastritis, periduodenitis and pericolitis. A patient with local peritonitis maintains a fixed posture for fear of severe pains likely to be caused by any movement, however slight. Pains felt during walking or jolty riding most likely indicate the presence of adhesive processes in the abdominal organs; pains before defecation are felt with a lesion of the descending part of the intestine and the sigmoid colon, while pains felt during defecation are evidence of a fissure, haemorrhoids, and an inflammatory process in the rectum. Pains in local and general peritonitis give rise to muscular resistance, rigidity of the abdominal muscles.

Meteorism (flatulence of the abdomen). Meteorism is the result of a large collection of gas in the intestine owing to its increased production by bacterial flora and carbohydrate food rich in cellular tissue (peas, cabbage, rye bread, sweets, whole milk, etc.), gas malabsorption in the intestine, as well as to venous stasis associated with a circulatory disturbance in the portal vein. The presence of a considerable amount of gas and liquid in the intestine causes a sensation of distention and difficult breathing. These symptoms are seen with inflammatory processes in the intestine but are also often encountered in neuropathic subjects.

Diarrhoea. This is a condition characterized by a frequent and loose stool. Diarrhoea is produced by a number of causes: (a) an increased intestinal peristalsis; (b) a disturbance in intestinal absorption of digestive juices; (c) secretory hyperfunction of the intestinal wall; (d) a disturbance in the chemical processes of digestion and the resultant irritation of the intestine.

With an increased intestinal peristalsis, there is no time enough for a complete digestion of food by the intestinal and pancreatic juices and part of the food is evacuated undigested. In the majority of cases, this kind of diarrhoea is associated with disorders in intestinal function due to a nervous shock, emotional turmoil, fear and sudden fright.

An increased peristalsis is also observed in cases of irritation of the mucous membrane by undigested food, and in endocrine disturbances—Basedow's and Addison's diseases.

Normally an adult person secretes about 5 litres of digestive juices in 24 hours, which upon completion of the digestive process are reabsorbed almost in full. In certain cases reabsorption of digestive juices is disturbed and results in frequent as well as copious evacuations of the bowels.

In an inflammation of the intestinal mucosa, the intestine contains secreted fluids rich in proteins and leucocytes, as well as pus and blood in cases of ulcerative processes occurring in its lower portions. An inflammatory secretion in the intestine is also noted with constipation when a retained faecal matter causes irritation of the mucous membrane and intensified secretion. An inflammatory diarrhoea is most commonly seen with diseases of the large intestine, while malabsorption and increased peristalsis occur with diseases of the small intestine.

Dyspeptic diarrhoea results from an insufficient digestive function of the stomach, the pancreas, the intestine, and the liver. A highly important role in the genesis of dyspepsia is played by a change in the intestinal flora. Dyspepsia may be fermentative, putrefactive, and fatty.

Fermentative dyspepsia is consequent upon a disturbance in the digestion of carbohydrates caused by ingestion of fermented food and sustained by overloading the intestine with foods abounding in carbohydrates (bread, coarse vegetables, cabbage, cucumbers, etc.). Acid products formed in the intestine cause an intensified peristalsis. Faecal matter has a putty consistency and is evacuated in quantity. Stools are of a pale-yellow colour, with an acid odour, and occur 3-4 times in 24 hours; they contain a large number of gas bubbles and undigested starch which stains blue on contact with iodine. The patient complains of abdominal distention; a splashing sound, rumbling, and tenderness are discovered on palpation of the abdomen.

The treatment of fermentative dyspepsia, particularly its severe forms, is begun with instituting 1-2 fasting days when the patient is given nothing but unsweetened tea. Then all foods containing coarse vegetable cellular tissue are withdrawn from the diet (rye bread, cabbage, potatoes, radishes, lettuce, etc.). The patient is maintained on a diet of butter, cottage cheese, eggs, biscuits, rusks and meat broth. After a considerable abatement of dyspeptic symptoms, the diet is widened to include carbohydrates and puréed vegetables. Calcium carbonate is prescribed in a dose of 0.5 g to be taken three times daily (formula No. 94).

Putrefactive dyspepsia is usually secondary to gastric achylia, hypofunction of the pancreas and the resulting disturbance of protein digestion. Decomposition processes normally occur only in the descending and the sigmoid colon, but in the case of putrefactive dyspepsia they extend to the higher parts of the intestine as far as the ileum. Putrefactive dyspepsia is also encountered with inflammatory processes in the intestine, when protein exudate undergoes putrefaction as well as after ingestion of spoiled protein foods. It is sustained and intensified by ingestion of meat and fish.

Stools in putrefactive dyspepsia are frequent, watery and copious. The faecal matter is of a dark colour, with an alkaline reaction and an extremely offensive odour. It contains a large amount of remains of undigested food and muscle fibres with retained striation. The patient is undernourished and emaciated.

In putrefactive dyspepsia, treatment is also begun with institution of 1-2 days of fasting or an exclusive apple diet, the patient being given 1-2 kg of sieved apples. The diet is later widened to include fruit soups, thin gruels, butter, and a small quantity of minced protein food added with caution. Milk and dairy products are forbidden for a long time, because they are likely to cause a severe exacerbation. It is recommended to prescribe hydrochloric acid with pepsin and pancreatin.

Fatty, or saponaceous, dyspepsia is caused by a rapid passage of food from the small into the large intestine, inadequate secretion of lipase, and a disturbance in the bile supply of the intestine. The faeces are of a pale-yellow colour, with a neutral or slightly alkaline reaction, and contain a significant amount of fatty acid crystals and soaps. In fatty dyspepsia, fats are at first withdrawn from the diet and later their amount is limited for a long period of time.

Anaphylactic, or allergic, diarrhoea is consequent upon the patient's intolerance of certain foods, such as strawberries, shellfish, etc. Ingestion by a hypersensitive subject of even a negligible quantity of such food causes diarrhoea, sometimes with manifestations of anaphylaxis on the skin (urticaria). In all cases of diarrhoea occurring only in one member of a family where this food was eaten. the physician should be alert to the possibility of allergy, especially when such a diarrhoea repeatedly troubled the sufferer earlier. Very similar to allergic diarrhoea is the condition due to the administration of certain medicines, for instance, iodine, bromide, etc.

Anaphylactic, or allergic, diarrhoea is prevented by withdrawal from the diet of foods to which the patient is hypersensitive.

Constipation (obstipatio). Constipation, or costiveness, is a prolonged pathologic retention of faecal matter in the intestine for over 48 hours, as well as a difficulty in evacuation due to a variety of causes.

Alimentary (food) constipation occurs after ingestion of readily digested and absorbed, even ground food (eggs, meat, fish, white bread, milk, etc.) which leaves little wastes. Its development is due to the absence of the required stimulants of intestinal peristalsis. Another contributing factor is a liquid deficit, which leads to an excessive dehydration and hardening of the faecal matter, interfering with the latter's passage through the intestinal tract.

Constipation may also be of nervous origin. Many persons may have their stools delayed in a state of dejection. Besides that, quite a few people feel an urge to empty the bowels under definite circumstances: in the morning on rising from bed or during a toilet, after a breakfast, etc. These conditioned reflexes are developed since childhood and their disturbance leads to constipation. This occurs in case of a change in living conditions, in travel, etc. Constipation of nervous origin includes all cases of retention of stools resulting from overstimulation of the vagus nerve under the influence of various diseases: gastric and duodenal ulcer, inflammation of the gallbladder, appendicitis, inflammation of the genitalia, etc. In addition, constipation may be secondary to organic lesions of the spinal cord and the brain. In certain cases, the cause of constipation is a congenital elongation or distention of the large intestine, endocrine diseases (myxoedema, etc.), narcotic intoxication, etc.

Sometimes constipation is associated with a history of inflammation of the large intestine (inflammatory constipation).

In many people, chronic constipation, only when running a protracted course, is manifested by numerous local and general symptoms: rumbling, splashing sounds in the intestine and a feeling of fullness, flatulence, and distention of the abdomen. Sometimes the patient is discomforted by a feeling of incomplete evacuation of the bowels, an unpleasant sensation and pain in the anus which become especially distressing in the presence of haemorrhoids. Among the general symptoms are headaches, dizziness, debility, early fatigue, and depression. All these symptoms rapidly pass when intestinal function has returned to normal. In chronic constipation, finger examination of the rectal ampulla is sometimes undertaken. This may reveal fissures, cysts, haemorrhoids, etc.

In constipation, the stools are dense in consistency and take the shape of small round masses resembling sheep droppings. Less frequently they are long and shaped as a ribbon or have round form resembling a lead pencil. In constipation joined by an inflammatory process in the large intestine, the faeces may be covered with mucus. The presence of fresh blood in the faecal matter is evidence of an affection of the lower part of the intestine. Bright-red blood and pus on the surface of the faeces is noted with ulcers of the large intestine and haemorrhoids.

The condition of the rectum and sigmoid colon is ascertained by means of rectoromanoscopy as well as x-rays. The contrast substance (barium) is introduced through the mouth, after which its passage along the intestinal tract is observed for a long time—for 6 to 96 hours. In addition, barium is also injected with an enema in order to examine the mucous membrane of the large intestine.

The treatment of constipation varies with its precipitating causes. A patient with alimentary constipation is given foods rich in coarse cellular tissue and substances of a chemical composition intensifying the intestinal peristalsis. Among such foods are rye bread, all vegetables, fruit, jams, honey, water-melons, melons, peaches, gooseberries, fresh kefir, vegetable oils, butter, etc. In constipation due to a nervous and mental unrest, sedatives (luminal, bromide, etc.) are helpful. Measures should be taken above all against those diseases (gastric and duodenal ulcer, etc.) which are a secondary cause of constipation.

ACUTE ENTERITIS (ENTERITIS ACUTA)

Aetiology and pathogenesis. An acute inflammation of the small intestine (enteritis) is mostly encountered in spring and autumn. It is due to an excess of carbohydrates (fruit, kidney beans, peas, etc.) in the diet, as well as to ingestion of spoiled protein food, mostly meat, and to food poisoning. Enteritis may also occur in pneumonia, influenza, Botkin's disease (infectious hepatitis), etc. Enteritis is caused by microorganisms which gain entry to foodstuffs when the latter are contaminated with infected water or infested by insects. This group of enteritides belong to nonspecific infectious enteritis.

Enteritis may also be secondary to an acute infectious disease with a predominant affection of the intestine (typhoid fever, paratyphoids, cholera). This group belongs to specific infectious enteritides.

Enteritis may also be produced by the action of toxic substances, in particular arsenic and mercury, certain industrial poisons, and a number of drugs (iodine, salicylates, adonis), as well as by overheating, burns, and chilling.

This disease may also result from hypersensitivity to strawberries, raspberries, and shellfish (alimentary idiosyncrasy).

Especially susceptible to enteritis are persons with secretory insufficiency of the stomach and the pancreas, debilitated subjects, and those with a deranged nervous system.

Enteritis rarely occurs as an isolated disease, and is commonly concomitant with an affection of the stomach (gastroenteritis) and the large intestine (entrocolitis).

Pathologic anatomy. Most commonly, enteritis develops as a catarrhal desease characterized by hyperaemia and oedema of the mucous membrane of the small intestine with punctate haemorrhages in the folds of Peyer's patches. In follicular enteritis the follicles are swollen. Enteritis is accompanied by a serous or fibrinous effusion, secretion of mucus, and desquamation of epithelial cells. This disease is rarely purulent and very seldom progresses to gangrene, with a possible perforation of the intestinal wall. Gangrenous enteritis develops in emaciated subjects with toxaemia and in persons with alimentary dystrophy.

Clinical picture. The clinical features and course of the disease depend on its aetiology and the reactivity of the body, therefore, in mild cases the patient feels little change in his general condition. In severe forms of acute enteritis, however, the damage to health is quite considerable. The disease is acute in onset, with loose stools recurring 5 to 10 times in 24 hours, pains, anorexia, nausea, sometimes repeated vomiting, at first of remnants of food, later of mucus with an admixture of bile, and gurgling and rumbling sounds within the abdomen. The pains are paroxysmal, are localized about the navel, and grow worse before evacuation of the bowels. Rumbling, gurgles, and pains mitigated after a stool again become more intense until another discharge of faeces. The stools at first have the consistency of putty, then become liquid and acquire an offensive odour. In view of their large content of unchanged bile the faeces are yellow or yellowish green; in choleraic enteritis the stools resemble rice-water. The stools in enteritis contain a large amount of mucus, muscle fibres, grains of starch, and drops of fat.

The patient is pale, with dry lips, sunken eyes, a dry and coated tongue, a distended abdomen; pain is felt throughout the abdomen and the right iliac region. In severe cases, there is a considerable rise in temperature and dehydration of the body due to a frequent stool, sometimes attended with symptoms of vascular insufficiency. In this condition the pulse is quickened and small, the arterial pressure drops, and the amount of urine voided is diminished. The urine sometimes contains erythrocytes and protein. The blood grows thicker and the erythrocytes grow in number. In severe enteritis, there are convulsions in the extremities and muscle pains due to a common salt deficiency in the body.

In the majority of cases, the disease ends in complete recovery; recurrent enteritis, however, may result in protein and vitamin deficiency and emaciation.

Treatment. A patient with acute enteritis should be confined to bed. If the disease has been caused by eating spoiled food, purgatives are administered: 1-2 tablespoonfuls of castor oil or 20-25 g of effervescent magnesium sulphate in half-a-glassful of water. In all cases of enteritis, sulpha drugs such as disulphan, sulgine, (sulphanilguanidine) and phthalazole (phthalyl sulphathiazole) in a dose of 4-6 g in 24 hours and antibiotics, such as levomycetin, biomycin, terramycin, tetracycline, etc., are helpful. Hot-water bottles, electric heaters, and compresses are applied to the abdomen. Pains are relieved with belladonna in combination with papaverine (formula No. 90). Carbon preparations are administered against meteorism (formula No. 80).

The patient must be strictly enjoined to eat only bland food during 3-5 days. He is allowed to have unsweetened tea, white rusks, rice water, mucilaginous soups with butter, puréed lean meat and fruit, and thin bilberry jelly. Food must be given several times a day in small portions and warm. Foods of coarse cellular tissue, as well as smoked foods, cold beverages, milk, seasonings, and icecream are forbidden.

CHRONIC ENTERITIS (ENTERITIS CHRONICA)

Chronic enteritis, nontropical sprue, idiopathic steatorrhoea are names of one and the same ailment—a chronic inflammatory degenerative affection of the small intestine. Chronic enteritis was for the first time distinguished as an independent disease by V. Obraztsov and A. Ostroumov.

Aetiology and pathogenesis. Enteritis is due to a variety of causes. It is commonly infectious in origin and occurs in bacillary dysentery, typhoid fever and paratyphoid fever, less frequently in influenza, pneumonia, and sepsis (nonspecific infection). Infectious enteritis shows the highest incidence in case of infection antedated by protein and vitamin deficiency, progressive dystrophy and metabolic disturbances. A hot climate may also be a contributing factor.

Alimentary factors play a significant role in the development of chronic enteritis. This is especially frequently seen in secretory insufficiency of the stomach and pancreas, in liver diseases, after ingestion of an excessive amount of carbohydrates (fruit rich in sugar, sometimes undergoing fermentation, vegetables, rice, etc.), and when the diet is low in proteins, particularly in a hot climate. In the intestine, carbohydrates undergo fermentation and methane decomposition, fermentative and putrefactive processes developing in the small intestine. Insufficiency of proteins and vitamins, especially those of the B complex, in the diet impairs the motor function of the small intestine, reduces the latter's capacity for absorption of proteins and vitamins in particular, resulting in a further aggravation of the pathologic process in the small intestine.

Chronic enteritis may be due to infestation by helminths and protozoa organisms. Helminths cause a mechanical and chemical damage to the intestine with products of their vital activity, and produce an allergic effect by sensitizing the intestine with substances which they discharge. Toxic enteritis is due to industrial poisons, arsenic, lead, phosphorus, as well as to a prolonged drug therapy (with salicylates, digitalis preparations, adonis, etc.).

Pathologic anatomy. There is distention and elongation of the small intestine, atrophic changes in the mucous membrane and lesions in all coats of the intestinal wall. The villi are smoothed out, indurated and thickened; they may be shortened or absent altogether, with a resulting disturbance in their absorptive and contractile activity. At the same time, signs of degeneration are noted in the nerve cells of the intestine, Meissner's and Auerbach's ganglia, and also in the lymphatic system which has an extremely adverse effect on the absorption of fats.

Clinical picture. The clinical manifestations vary with the stages of chronic enteritis. The most typical symptoms are pains and distention in the middle part of the abdomen with an increased passage of gases in the latter half of the day, when the digestive process is at its height. Pains are sometimes very severe. There are rumbling and splashing sounds in the caecum due to an accumulation of undigested food particles and profuse secretion. Diarrhoea is an important, although not a regular, symptom of chronic enteritis. Stools occur from 8 to 10 times in 24 hours. There is a reduction in protein absorption (60-70 per cent of the amount ingested, instead of the normal 90-95 per cent). The protein level of the blood serum is diminished to 3-4 g/100 ml with a simultaneous reduction in albumins and increase in coarsely dispersed globulins. A protein deficiency entails a disturbance in the normal enzymatic function of the gastrointestinal tract with an ensuring impairment of food digestion and absorption.

The assimilation of fats is reduced to 30 per cent. A disturbance in fat absorption is evidenced not only by steatorrhoea but also by the fact that the amount of fat in the blood is increased insubstantially, if at all, in severe enteritis after the administration of various fatty test breakfasts. Malabsorption of fat is attended with a considerable diminution in the phospholipid and cholesterol content of the blood. The absorption of carbohydrates is impaired relatively slightly, the blood glucose content of the blood in such patients, however, is not higher than 60-80 mg/100 ml instead of the normal 80-120 mg/100 ml. A disturbance in the water-salt metabolism is encountered only with severe forms of chronic enteritis. At the same time, the disturbance involves the electrolyte metabolism due to a considerable loss of electrolytes, potassium in particular. In inflammatory degeneration of the small intestine there is vitamin deficiency due to a disturbance in the processes of absorption and vitamin synthesis in the intestine. It is known that folic acid, vitamins B_2, B_1, B_6, and K, biotin and nicotinic acid, are synthesized by the intestinal flora. In chronic enteritis, bacteria normally inhabiting the small intestine are suppressed, while the growth of other microorganisms depending on vitamins for their vital activity becomes more intensive. Vitamin deficiency entails the appearance of a number of new symptoms. A vitamin B_{12} deficiency gives rise to vitamin B_{12}-folic acid deficiency macrocytic anaemia. Malabsorption of fat is concurrent with that of fat-soluble vitamins A, B, and K. Vitamin K insufficiency results in a diminution of the prothrombin content of the blood and in proneness to bleeding. There is a steep reduction in nicotinic acid synthesis with an ensuing persistent and exhausting diarrhoea, glossitis (inflammation of the tongue), burning and redness of the tongue (raspberry tongue), atrophy of the papillae (polished tongue), erosions and fissures. There is also pigmentation of the skin in exposed places.

The calcium and phosphorus metabolism is disturbed. The sharp reduction in the calcium and phosphorus content of the blood is responsible for brittleness of the bones, osteoporosis, skeletal deformities, and osteomalacia due to a reduction in the calcium compounds in bones. A disturbance in calcium metabolism occurs simultaneously with that of fat absorption in the intestine, inasmuch as unabsorbed fatty acids form insoluble calcium soaps. A factor contributing to osteoporosis is also vitamin D deficiency. This vitamin regulates calcium and phosphorus absorption in the intestine. There is a disturbance in iron absorption which, along with vitamin B_{12} deficiency, is responisble for anaemia.

In chronic enteritis, the function of the endocrine glands is also impaired. Adrenal hypofunction entails general debility, arterial hypotension, and a grey discoloration of the face and hands (addisonism). Patients with a disturbed pituitary function complain of intense thirst, while the specific gravity of urine becomes low (like in diabetes insipidus). When enteritis is joined by colitis, diarrhoea makes its appearance, whereas in enteritis concomitant with hyposecretion of the stomach, intestinal dyspepsia is joined by gastric dyspepsia (nausea, vomiting, pains in the epigastrium, etc.). Cases are frequent of chronic gastritis and enteritis being due to one and the same cause.

Sometimes gastritis is a precursor of enteritis, especially in the case of secretory insufficiency, when the bactericidal properties of the gastric juice are reduced to naught and favourable conditions are provided for the development of inflammatory lesions in the small intestine. In chronic enteritis, the disease process may extend from the small intestine to the bile ducts and liver. Changes in the liver may be brought about by a disorder in protein metabolism, as is the case in chronic enteritis. In such cases the liver may suffer fatty infiltration and cirrhosis.

Examination of the faeces (Coprology). The stools in isolated enteritis are of normal shape and consistency. The specific signs of faecal discharges in chronic enteritis are a grey-yellowish or clay colour, an increase in the evacuations to 1-2 kg in 24 hours due to a disturbance in digestion and absorption, and the presence in the faeces of a considerable amount of undigested muscle fibres (creatorrhoea), soaps and neutral fats (steatorrhoea). The faecal matter is malodorous and contains visible remains of food, sometimes pieces of meat. This symptom is important for the diagnosis of chronic enteritis only in the absence of pancreatitis and secretory insufficiency of the stomach. The faeces may be lustrous, fatty, with a crust readily forming on their surface. The faecal matter slowly passing through the large intestine are darkened and shaped. The presence of a large amount of fats and soaps lends the faeces a grey clayish colour and a viscid, sticky consistency. Neutral fats, fatty acids and their salts (soaps) are evacuated with the faeces.

Treatment. Among the therapeutic measures, dietotherapy is the most important. A well-balanced diet of high caloric value is prescribed. The content of proteins, mainly of animal origin (meat, fish, cottage cheese, eggs) should not be less than 120-150 g, of fats, 50-60 g. The intake of carbohydrates, particularly those rich in starch and cellulose, should be restricted because of the fermentation they are known to cause. Carbohydrates, especially raw vegetables, legumes, fruit, prunes, berries used for wines, etc., in addition to fermentation, increase the motor and secretory activity of the small intestine. The diet must be rich in mineral salts, calcium and phosphorus (cheese) in particular. Fruit juices containing tanning substances (bilberries, quinces, bears, cornelian cherries, etc.) and common salt, if no oedema is present, are recommended. During an exacerbation, the patient is given puréed food and is enjoined to abstain from intakes of foods containing coarse vegetable and animal cellular tissue, as well as from milk, rye bread, pickles, smoked foods, cold dishes, and aerated beverages. All foods containing extractive substances and roasted foods are forbidden. The patient must have his meals in small portions. In enteritis concurrent with diseases of the liver, gallbladder, and pancreas, appropriate amendments should be made in the diet. Parenteral administrations of vitamins B_1 (10-20 mg daily), C (100 mg), nicotinic acid (20-30 mg), riboflavin (0.005 g two times daily), B_{12} (200 γ every other day during 10-15 days), and folic acid (0.02 g three times daily) are prescribed. For severe forms of the disease, intravenous infusions of glucose, hypertonic solutions of sodium chloride, and calcium chloride are helpful. Remedies recommended for parenteral administration are protein nutrients, such as blood serum, blood plasma, hydrolysates, and amino acid mixtures (per os or subcutaneously in a dose of 1 litre and more). In case of a severe dehydration of the body a physiologic salt solution is injected subcutaneously. For diarrhoea, antibiotics (levomycetin, biomycin), sulpha drugs (phthalazole, sulgine) pancreatin (1 g given 3-4 times in 24 hours), and bismuth nitrate and other astringents are administered. In anaemia, iron preparations in combination with ascorbic acid, haemostimulin *, and other medicines are efficacious. Prednisone and prednisolone are also administered in order to improve the patient's general condition: whet the appetite, increase the body weight, and reduce the frequency of stools and the fat content of the faeces.

SPRUE (SPRHUE)

Sprue is a disease running its course with a persistent fermentative diarrhoea, glossitis and anaemia. It takes its name from the Dutch *sprew*—foam.

Aetiology and pathogenesis. A complex of exogenous and endogenous factors plays a role in the origination of sprue: undermined health as a result of precursory diseases, nervous and mental strain, malnutrition, mainly due to a carbohydrate deficiency, a hot climate, and pregnancy and lactation when the vitamin and protein reserves of the body are exhausted and considerable abnormalities occur in the endocrine system. Sprue often develops in the wake of dysentery. All these factors inhibit the function of the digestive glands and cause a disturbance in intestinal digestion and absorption.

Attempts have been made to associate this disease with a certain fungus; the reproduction of yeast fungi in the intestine, however, depends on an unbalanced diet with prevalence of carbohydrates. Fungal flora causes dyspepsia with a persistent diarrhoea, progressive vitamin deficiency and emaciation. Sprue is attended with severe anaemia due to a disturbance in the formation and absorption of folic acid in the intestine.

Pathologic anatomy. Pathoanatomic changes in the walls of the small intestines are characterized by lymphocytic infiltration, thinning and sometimes by ulceration of the mucous membrane. Degenerative and atrophic changes in the parenchymal organs and hyperplasia of the bone marrow are encountered.

Clinical picture. The cardinal symptoms are as follows: persistent fermentative enteritis, glossitis, anaemia, affection of the endocrine and peripheral nervous systems and emaciation.

The patient complains of severe abdominal distention, thirst, an intensive formation of gases, a feeling of heaviness or pressure in the abdomen, pains in the lower abdomen, and loud rumbling and gurgling sounds. Distention and rumbling are usually followed by profuse, watery, light-coloured and malodorous stools recurring 5-6 times in 24 hours. The faeces are whitish from a large amount of unabsorbed fat, frothy, and contain many fatty acids and soaps, connective tissue, undigested muscle fibres, and yeast fungi. In enteritis complicated by colitis, the patient complains of frequent tenesmus, while the faeces contain mucus in quantity.

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* Haemostimulin—preparation containing cattle blood, iron lactate, copper sulphate and glucose (*Ed.*).

Prolonged enteritis unamenable to therapy with an ensuing malabsorption of nutritive and energy-producing substances in the intestine results in a severe emaciation and dehydration of the body, cessation of perspiration, secondary dystrophy and polyavitaminosis (mainly a lack of the vitamin B complex, as well as vitamins C and D).

Sprue causes atrophy of the tongue papillae (glossitis), oesophagitis, atrophic changes in the thyroid, parathyroid and sexual glands, a lesion of the adrenals, sometimes hypothyrosis, hypocalcaemia, hypotension and adynamia. There is amenorrhoea in women and impotence in men.

In the majority of cases, there is organic, less often functional, achylia and anaemia which is mainly due to a deficiency in folic acid and vitamin B_{12}.

Sprue runs its course in a mild, moderately severe and severe forms. The latter is accompanied by persistent diarrhoea, cachexia, sometimes in combination with oedema and considerable nervous and mental unrest. The patient is forgetful and sleepy. In women sprue is encountered in the period of pregnancy, lactation and menopause.

Prognosis. When neglected, the disease is highly resistant to therapy and often gives relapses, particularly in summer. Sometimes, it is complicated by tuberculosis and other infections. A cerebral derangement in the patient should be regarded as a formidable complication dimming his prospects of recovery.

Treatment. The diet must be nutritious, containing mainly animal proteins and a moderate quantity of fats and carbohydrates. It should include fresh cottage cheese, steamed lean meat, lean cheese, kefir, eggs, butter, and walnuts. The intake of liquid must be restricted, since its absorption in the intestine is disturbed by inflammatory changes. Of fruit juices, only that of pomegranate, which contains tanning matter, is recommended. With amelioration in the patient's general condition and appearance of shaped stools the diet may be widened to vegetable purée, fruit, semolina and rice porridge, and sugar. However, one should abstain from drinking whole milk for a long time.

The recommended drugs are cod-liver oil, calcium (formula No. 54), hydrochloric acid in large doses, pancreatin, nicotinic acid, and bismuth. Injections of vitamin B_{12} in doses of 50-100 γ every other day are also prescribed.

For severe dystrophy or anaemia, transfusion of whole blood and erythrocyte mass is indicated. Fresh forms of sprue readily respond to complex therapy and resolve in complete recovery. In case of a relapse in a hot climate, the patient is advised to move to a temperate climate for summer.

ACUTE COLITIS (COLITIS ACUTA)

Colitis (Gr. *kolon*—large intestine) is an inflammation of the mucous membrane of the colon, which may be acute or chronic.

Aetiology and pathogenesis. Acute colitis is commonly a sequel to bacterial dysentery, less frequently to amoebiasis, balantidiasis, lambliasis, etc. As a concomitant disease, it is encountered in persons eating food infested by *Salmonella* organisms, staphylococci, streptococci, bacteria of the genus *Proteus*, *E. coli Paracolobactrum* organisms. It occurs in typhoid and paratyphoid fevers, in measles, influenza, and septic conditions. Sometimes, acute colitis develops after ingestion of abundant, poorly cooked and irrigating, mainly carbohydrate foods, as well as after administration of certain drugs to which the patient is hypersensitive. Colitis may be due to poisoning with mercury, arsenic and benzene, and to uraemia in which toxic matter is eliminated into the large intestine.

Infectious colitis with a simultaneous affection of other parts of the digestive tract in addition to the colon is known as gastroenterocolitis.

Pathologic anatomy. The mucous membrane is red, oedematous, and covered with mucus and pus. In severe cases of acute colitis due to dysentery, mercury poisoning and uraemia a diphtheroid inflammation (fibrinous exudation in the wall) and gangrene with subsequent perforation may be observed. The entire large intestine or some of its segments are involved.

Clinical picture. Acute colitis commonly coexists with gastritis and enteritis. The clinical picture varies with the aetiology and localization of the disease process. The typical symptoms are as follows: an acute onset with diarrhoea, fever, and colicky pains localized in various segments of the colon. The most intense pains felt as colics and gripes along the course of the large intestine, as well as tenesmus occur with a disease process localized to the left half of the colon. Stools are frequent, recurring up to 20 times and more in 24 hours, at first malodorous, later with mucus and an admixture of pus, as well as mucosal scraps. Fever rises to 38-39°C, the tongue is dry and coated, the appetite is absent, and the patient complains of headaches.

A somewhat different course is taken by acute colitis mainly localized to the right half of the colon. The disease is less acute in onset, pains are less severe and radiate to the small of the back and to the right thigh. Stools recur not more than 10 times in 24 hours and without tenesmus, the faeces are liquid and with an offensive odour but contain no visible mucus, blood or pus since these admixtures undergo digestion in the right half of the colon. The temperature is mainly subfebrile; however, severe general intoxication (lassitude, headaches) is present. There is neutrophilic leucocytosis and an elevated ESR. The abdomen is distended and the colon is tender on palpation. In case of a lesion in the caecum a painful fibrous band is palpable in the right iliac region. An identical band is palpable in the left iliac region in case of left-side colitis.

Acute colitis lasts from a few days to a fortnight and resolves in recovery or becomes chronic with a proneness to relapses. It may also give rise to various complications, such as stenosis of the sigmoid flexure, a purulent or gangrenous inflammation with perforation of the colon, etc.

Treatment consists in efforts to eliminate the cause of the disease. Regardless of the aetiology, however, the following measures are indispensable: (a) complete bed rest and, in the event of dysentery, isolation of the patient; (b) application of hot-water bottles, electric heaters, and hot compresses to the abdomen; (c) an adequately nutritive but bland diet with elimination of mechanical and chemical irritants as well as drug therapy.

In the early days of the disease the patient is given only lukewarm unsweetened tea, and from the third day, mucilaginous soups. After cessation of diarrhoea, the diet is supplemented with meat and fish broths, minced meat and vegetable purée and floury dishes, and carrot and fruit juices. The intake of fresh unskimmed milk, rye bread, fancy bread, sausages, legumes, spices, tinned foods, appetizers, raw vegetables and fruit is forbidden. The diet must contain 120-150 g of proteins, 60-70 g of fats, 300-400 g of carbohydrates, 10 g of common salt, and vitamins of the B, C, and A groups. A single administration of purgatives is prescribed. In severe cases, purgatives such as a 2 per cent magnesium sulphate or sodium solution are to be taken during one or two days in fractional doses totalling 1 litre in 24 hours. It is mandatory to administer sulpha drugs which are slowly absorbed from the intestine—disulphan, sulgine, phthalazole—in doses of 1 g to be taken 5-6 times daily during a period of 5-7 days. Sulpha drugs are combined with broad spectrum antibiotics, such as levomycetin (synthomycin) to be taken per os in a dose of 0.5 g three times daily, streptomycin, biomycin, as well as tetracycline.

Belladonna suppositories or microenemas of antipyrine (0.5 g per 10 ml of water after a cleansing enema) are administered for a severe tenesmus. In dehydration of the body, a physiologic salt solution or a 5 per cent glucose solution in a dose of 500 ml is injected intravenously or subcutaneously. Subcutaneous injections of caffeine, cardiazol, cordiamine, etc., are employed in cardiac insufficiency. In amoebic colitis (amoebiasis) emetine sulphate (formula No. 169) in a dose of 0.05 g is injected subcutaneously 2-3 times daily during a period of 5-6 days, and this treatment is repeated 2-3 times at 5-day intervals.

CHRONIC COLITIS (COLITIS CHRONICA)

Aetiology and pathogenesis. The most frequent cause of chronic colitis is dysentery which may leave a disturbance in intestinal function upon recovery. Chronic colitis results from food poisoning, amoebiasis, balantidiasis, lambliasis, infestation by helminths, as well as from a systematic overeating or a prolonged bland diet. Other contributing factors are protein and vitamin deficiency in food, abuse of purgatives and enemas. Colitis is often secondary to a persistent constipation of functional origin, when congested faecal matter causes a mechanical and chemical irritation of the mucosa of the colon. Among the predisposing causes of chronic colitis are chronic gastritis with secretory insufficiency, diseases of the pancreas, liver and small intestine, congenital and acquired anomalies in the colon (megasigmoid, dolichosigmoid), adhesions in the intestine as well as derangements in the central nervous system. An exacerbation of chronic colitis is often seen after recovery from influenza, tonsillitis, and in chilling, nervous shock and dietary abnormalities.

Pathologic anatomy. Colitis may be extended through the entire length of the large intestine (pancolitis) or it may be segmentary when the disease process is mainly confined to the caecum and ascending colon (typhlocolitis), the sigmoid flexure and rectum (proctosigmoiditis, rectitis), and less frequently to the transverse colon (transversitis). The inflammatory process may have the following forms: (a) catarrhal; (b) fibrous; (c) necrotizing; (d) ulcerative. In a catarrhal inflammation, the mucosa is oedematous and hyperaemic (congested), at places covered with mucus and erosions. Membranous colitis (mucomembranous colitis) belongs to the same group. A fibrinous inflammation of the large intestine is seen with dysentery, arsenic and mercury poisoning, and in uraemia. In chronic ulcerative colitis, the mucosal surface is covered with ulcers which invade the submucosal layer and sometimes penetrate into the depth of the intestinal wall. The latter is thickened, sometimes atrophied, while the mucosal surface is pale and swollen.

Clinical picture. The clinical features of chronic colitis vary with the localization of the pathologic process and the presence of concomitant diseases. The typical signs are frequent liquid putty stools with an admixture of mucus, sometimes pus and blood, and with signs of putrefactive or fermentative dyspepsia. Sometimes diarrhoea makes its appearance before or shortly after a meal, early in the morning, or in the daytime at regular intervals. In certain cases, paroxysms of rectal urgency follow one another in rapid succession. This occurs most commonly when the disease process is localized to the descending colon. In some cases there is tenesmus with a discharge of mucus and gases only.

A characteristic feature of chronic colitis is alternation of diarrhoea with constipation. The former is consequent upon a dietary negligence, such as eating fatty food, drinking cold water, milk, etc. During constipation the patient feels much better than during a flux, since he is emaciated by the latter's frequent recurrence.

Examination reveals abdominal distention and tenderness on palpation, induration of the walls of the colon, and loud rumbling and gurgling sounds in the abdomen. In an affection of the left half of the colon pains are localized to the left iliac region, radiating to the small of the back, left leg and buttock. There is constipation followed by diarrhoea. The sigmoid flexure is thickened, spastically contracted, tender, knobby and limited in mobility. In inflammation of the rectum, the patient complains of a burning sensation in the anus, tenesmus, and pains during defecation, which become particularly intense in the presence of fissures in the rectum. In such cases, rectoromanoscopy reveals redness and oedema as well as ulceration of the mucous membrane. Upon extension of the inflammatory process from the colon to the surrounding tissue (pericolitis) pains grow worse on physical exertion and in jolty riding.

With a disease process mainly localized to the right half of the large intestine (typhlocolitis), the patient complains of pains in the right half of the abdomen radiating to the small of the back. There is retention of bowel evacuation, although liquid stools are seen sometimes, the intestine is distended, tender on palpation, with rumbling and gurgling sounds in the abdomen. The liver and bile ducts are most frequently damaged. With a disease process extending from the mucous membrane to the surrounding cellular tissue (perityphlitis) in the stage of exacerbation Shchetkin-Blumberg's sign is positive. Typhlocolitis must be differentiated from appendicitis and tuberculosis of the ileocaecal region, which produce a similar clinical picture.

Chronic colitis of dysenteric origin is commonly localized to the left half of the large intestine. In exacerbations the faeces contain mucus rich in neutrophilic leucocytes and intestinal epithelial cells. Triboulet's test (for insoluble protein) is positive. Rectoromanoscopy reveals the presence of catarrhal, erosive and even ulcerative colitis.

The diagnosis of chronic colitis of dysenteric aetiology is verified by isolation of dysentery bacilli or a positive serologic test.

Protozoan colitis is prevalent in tropical and subtropical countries as well as in localities with a moderate climate. Intestinal lambliasis is the most frequent. *Lamblia* parasites inhabit the upper parts of the intestine and often cause duodenitis and enterocolitis. The principal symptoms of colitis due to *Lamblia* organisms are transient diarrhoea and the presence of the parasites or their cysts in the faeces.

The inflammatory products found in the stools of this disease are scant.

CHRONIC ULCERATIVE COLITIS OF UNRECOGNIZED AETIOLOGY

Ulcerative colitis, otherwise known as chronic nonspecific ulcerative colitis or thromboulcerative colitis, is common in tropical countries but is also encountered in a temperate climate.

Aetiology and pathogenesis. Various theories have been advanced to explain the aetiology of ulcerative colitis (bacterial theory, virus theory, etc.). The most reasonable is the allergic theory which attributes the disease to immuno-allergic, neuro-endocrine, metabolic and other causes.

Pathologic anatomy. There are ulcerations, superficial or deep, eroding the muscular and serous coats and frequently resulting in perforation of the intestine. Most commonly the disease process is localized to the sigmoid flexure and rectum. The mucous membrane is covered with pus, mucus, blood, sometimes with multiple granulomas and polyps.

Clinical picture. Ulcerative colitis is encountered at all ages, more often in women than in men. The disease usually marks its onset with a copious mucosanguineous or pyosanguineous discharge from the rectum, tenesmus, frequent stools (7 to 20 and more in 24 hours), abdominal pains, lassitude and weight loss. Ulcerative colitis may run a malignant or benign course.

The malignant form is relatively rare, runs a violent course with frequent stools (up to 60-70 in 24 hours), a high-grade fever, profuse intestinal bleeding, anaemia, neutrophilic leucocytosis, emaciation, and a disturbed water-salt balance. The faeces contain mucus, blood and pus, thoroughly intermixed. The disease lasts not longer than 1-2 weeks and often ends in death, in particular from complications—massive haemorrhage or perforation of the intestine.

The benign form runs its course with abdominal pains felt before defecation, sometimes with tenesmus, bloody diarrhoea with an admixture of mucus and pus, and fever. The patient's condition is satisfactory, but it grows worse after each relapse which usually follows a remission. Relapses mostly occur in spring or autumn and are, as a rule, provoked by various infections (influenza, tonsillitis, etc.).

Rectoromanoscopy reveals haemorrhages, erosions, and small ulcers in the rectum and caecum. Ulcerative colitis involves a number of changes in the visceral organs and systems: gastric achylia, myocardial dystrophy, a disturbance in the function of the liver, etc. Hypochromic anaemia, neutrophilic leucocytosis with an increase in the number of rod neutrophils and an elevated ESR are also encountered. The disease is accompanied by hypoproteinaemia.

X-rays reveal a narrowing of the intestinal tube, a tortuous mucosal relief, cicatricial-ulcerative strictures of the rectum and sigmoid flexure, sometimes multiple polypous proliferations.

Complications. Ulcerative colitis is often responsible for intestinal haemorrhages, strictures and obstruction of the intestine, paraproctitis, haemorrhoids, anal fissures and fistulas, liver cirrhosis, amyloidosis, etc.

Differential diagnosis. Ulcerative colitis must be differentiated from bacterial dysentery and amoebiasis. Detection of the causal agents of these two infections is of decisive importance for the diagnosis.

Treatment consists of a complex of therapeutic procedures. In the acute stage, physical and mental rest, a bland diet of high caloric value, with elimination of mechanical and chemical irritants and rich in nutritious proteins (120-150 g in 24 hours) are necessary. The intake of carbohydrates is limited if fermentation is present, pickles and spicy foods are forbidden, and the ingestion of fats is restricted to 50 g in 24 hours. The diet is enriched with vitamins of the B-, C- and A-groups. There must be 4-5 meals daily, with total abstinence from milk and dairy products.

Drug therapy is with sulpha drugs and antibiotics (sulgine, disulphan, tetracycline, terramycin). Among the remedies widely applied during the past few years are ganglionic blocking drugs, steroid hormones (prednisone, prednisolone) in doses of 30-60 mg daily, ACTH in doses of 20-30 U, and transfusions of blood and plasma.

MUCOUS COLIC (COLICA MUCOSA)

Mucous colic is also known as spastic colon myxomembranous or mucous colitis (colitis spastica, colitis membranacea).

Aetiology and pathogenesis. Mucous colic is a neurosis of the large intestine manifested by a disturbance in its secretory and motor functions. Lodged within the branch of the vagus nerve supplying the intestine are the secretory fibres the stimulation of which causes mucus hypersecretion, as well as the motor fibres whose stimulation gives rise to an intensified peristalsis and an increase in the tone of the large intestine. In addition, there are vasodilating fibres the stimulation of which produces hyperaemia of the intestinal wall.

Several theories have been offered to explain the causes of mucous colic: infectious-inflammatory, hepatic, thyrogenous, allergic and neurogenous. Most of the contemporary authors consider mucous colic a neuro-allergic disease like bronchial asthma (the fact that it most frequently occurs in patients with bronchial asthma stands in favour of this view), gastric and duodenal ulcer, gastric hypersecretion, and hypertensive vascular disease, i.e., maladies which are basically due to a heightened excitability of the nervous system, particularly the vegetative system. These characteristic features of the clinical course of mucous colic have induced many authors to regard it as intestinal asthma. Mucous colic may be complicated by secondary inflammatory changes in the large intestine. The immediate onset of attacks of mucous colic in the wake of nervous and emotional upset as well as a reflex stimulation in pathologic conditions of the abdominal organs is evidence of an important role played by the nervous system in the genesis of this disease.

Clinical picture. Mucous colic is often preceded by a feeling of fulness and distention in the abdomen and of gurgling in the bowels. An attack of colic occurs as intense paroxysmal diffuse pains in the abdomen, which are localized mainly to the left iliac region (descending colon), less frequently to the right iliac region (ascending colon), or to the middle of the abdomen (transverse colon). Paroxysmal pains of a spasmatic character occur suddenly, usually in connection with a nervous overstrain, and are accompanied by pallor of the face, sometimes an intense anxiety followed by a discharge of greyish-white membranous shreds shaped like tapeworms. Paroxysmal pains, however, are not inevitably accompanied by a mucus discharge.

Palpation of the abdomen reveals tenderness along the course of the large intestine, which is spastically contracted all along its length, but mostly in the region of the descending and sigmoid colon. An attack of pain is attended with retention of stool. Mucous colic may run its course like dysentery, i.e., with frequent stools containing mucus and a rise in temperature. Sometimes, it may resemble a typhoid infection but usually it is characterized by intense colicky pains, vomiting, constipation, fever, dizziness, palpitation, and pallor of the face. An attack lasts 30 minutes to several hours or days and ends in a discharge of 100-500 g of white mucous masses shaped like tapeworms, plates and tubes resembling an exact cast of the large intestine with its transverse and longitudinal folds. The faeces are sometimes enveloped by mucus or covered with thin membranous shreds. Seen under a microscope mucus contains a small number of desquamated epithelial cells, leucocytes with predominance of eosinophils, and Charcot-Leyden crystals. A characteristic feature of mucous colic is eosinophilia in the blood, which rises to 10-30 per cent in a period of remission. During an attack, the number of eosinophils in the blood is normal, but is considerably increased in the mucus.

Relapses of mucous colic alternate with relatively quiet periods. Attacks may recur after weeks, months and even after a year. In the intervals between attacks the patient suffers from chronic constipation with predominance of spastic symptoms. Sometimes, pains become especially intense 5-6 hours after a meal, which corresponds to the entry of food into the proximal segment of the large intestine. Mucus may be discharged with faeces not only during an attack of pains but also in the period of remission. Emaciation and anaemia develop in protracted cases of the disease. In addition, various complications come on the stage, such as pyelitis, nephrolithiasis, cholelithiasis, etc.

The *diagnosis* is established on the basis of characteristic attacks with a discharge of membranous shreds and a heightened nervousness. A differential diagnosis is made between mucous colic and conditions attended with excretion of mucus due to a prolonged administration of purgatives and enemas, appendicitis, proctitis, sigmoiditis, diseases of the gallbladder, kidneys, and female genitalia. During an acute attack, one is obliged to consider the possibility of pyelitis and cystitis which produce changes in the urine. A lead colic differs from a mucous colic by the presence of hypertension and a lead line on the gums.

Treatment consists in complete bed rest, application of heat to the abdomen (compresses, hot-water bottles, and fomentations), while the spasm is eliminated by a subcutaneous injection of 0.5 ml of a 0.1 per cent atropine solution, 1 ml of a 1 per cent promedole solution, or by papaverine given per os 2-3 times daily. If there is mucus retention an enema of vegetable oil is recommended. In the periods of remission prolonged atropinization is prescribed. Constipation is treated by means of a bland diet, with elimination of mechanical irritants. Other remidies are a 5 per cent vitamin B_1 solution in a dose of 1 ml to be administered during a period of 15-20 days, a 0.1 per cent nicotinic acid solution in a daily dose of 2-3 ml, ACTH in a dose of 20-30 U daily, prednisone or prednisolone in a daily dose of 15-30 mg to be administered during 2-3 weeks, anallergin to be taken in a dose of 1 tablet 3-4 times daily, and dimedrol in a dose of 0.02 g 3 times daily.

The nervous system is fortified by means of baths, sports, and physical culture. It is important to cure inflammatory processes in the abdominal, urinary and genital organs.

CHRONIC ENTEROCOLITIS (ENTEROCOLITIS CHRONICA)

The aetiology of chronic enterocolitis is identical with that of chronic enteritis complicated by colitis. Chronic enterocolitis produces an irritation of the epithelial lining of the intestine, vascular hyperaemia, and changes in the mesenteric lymph nodes and interstitial tissue. The inflammatory process in the intestinal mucosa may extend as deep as the serous membrane and even the parietal peritoneum.

The *clinical picture* varies with the phase of the disease. At first, the patient complains of an abdominal discomfort and pains in the epigastrium and throughout the abdomen, which grow worse before and after defecation. In the majority of cases, chronic enterocolitis takes a protracted course lasting for months and sometimes years. There is progressive emaciation, anaemia, increased irritability, and a considerable loss of the capacity for work. At the same time, trophic disturbances make their appearance, such as atrophy of the tongue mucosa, alopecia, shedding of the nails and dryness of the skin, which are most probably due to a disturbance in the synthesis and absorption of vitamins, those of the B complex in particular.

During an objective study a slight tenderness is revealed on palpation in the umbilical region and in the sigmoid colon. Stools of a putty consistency recur 3-4 times in 24 hours. Less frequently, there is constipation alternating with diarrhoea, the former lasting longer than the latter. The stools contain a considerable amount of mucus thoroughly intermixed with faecal matter, the latter sometimes being covered with mucosal scraps if the inflammation has spread to the lower segments of the intestine. Frequent tenesmus is evidence of a rectal irritation. Microscopy of faecal matter reveals the presence of pus and undigested food particles, especially in a predominant affection of the small intestine. The faeces have an alkaline reaction and a putrid odour. The urine contains indican and urobilin. The treatment of chronic enterocolitis consists in complex and individualized measures varying with the aetiology of the disease, local lesions, the nature of the pathologic process, the clinical picture, prevalence of putrefactive or fermentative processes, the patient's general condition, complications, and the acuteness of the course of the disease. In a period of exacerbation complete bed rest is mandatory. The basic remedy in this period, just as during a remission, is dietotherapy. The diet must contain an increased amount of nutritious proteins causing a lipotropic effect and a normal amount of fat; the level of carbohydrates varies with the character of dyspepsia but coarse vegetable cellulose must be completely eliminated. The intake of common salt is slightly restricted. Drug therapy includes sulpha drugs, antibiotics, Chernokhvostov's vaccine, sera, hormone preparations and vitamins (B, B_1, B_3, PP, E). In the event of nutritional disturbance with a diminution of body resistance fractional transfusions of whole blood and plasma are helpful. For diarrhoea hydrochloric acid, pepsin and pancreatin are recommended.

APPENDICITIS

Appendicitis is an inflammation of the vermiform appendix of the caecum.

Aetiology and pathogenesis. It was formerly believed that inflammation of the vermiform appendix was due to faecaliths and foreign bodies—fragments of enamel from kitchen-ware, berry and fruit stones, and parasites. These mechanistic views, however, have proved to be unfounded because, firstly, wounds due to a mechanical injury heal readily; secondly, faecaliths are often encountered in normal appendixes also. Later, a theory of enterogenous infection was suggested, which attributes appendicitis to the bacterial flora of the intestine, mainly to *E. coli.* The latter becomes highly virulent in the presence of congestive phenomena in the cavity of the vermiform appendix, which are often observed in case of twisting of the appendix, stenosis of its cavity, and stasis in the caecum. According to the theory of enterogenous infection, appendicitis follows in the wake of inflammatory processes in the small intestine and caecum, since they are accompanied by an intensified reproduction of bacteria in the intestine, a considerable increase in their virulence and their penetration into the appendix wall. Efforts have also been made to associate appendicitis with the entry of microorganisms into the wall of the appendix with the blood flow from distant inflammatory foci (in the tonsils, gallbladder, etc.). However, blood cultures in acute appendicitis have proved to be sterile, while an intravenous injection of bacteria to rabbits has failed to induce appendicitis. These studies have disproved the theory of haematogenous infection of the vermiform appendix. Subsequently, a neurovascular theory was suggested to explain the aetiology of acute appendicitis. According to this theory, the pathologic process in the appendix results from a stimulation of the nervous system regulating the normal blood flow.

The latter theory is corroborated by the fact that in the presence of all clinical symptoms of acute appendicitis the appendix shows little change in the early hours of the disease in 20-30 per cent of cases. It should be noted in conclusion, that of all these theories the theory of enterogenous infection is the most reasonable. In the genesis of appendicitis great importance is attached to predisposing factors, such as chronic constipation causing congestion of faecal matter in the appendix, infectious diseases (influenza, German measles, scarlet fever, tonsillitis), septic condition, alimentary factors, in particular a diet abounding in meat, which is likely to cause an intensified reproduction of bacteria. Constitutional features of the structure of the vermiform appendix predisposing to its being readily infected also play a part.

Pathologic anatomy. Appendicitis may be acute or chronic. The former occurs in the following forms: catarrhal, purulent (phlegmonous), and perforative. The catarrhal form is characterized by a severe congestion of the vessels of the appendiceal serous coat and a thickening of its wall. The purulent, or phlegmonous, form is attended with pus formation in the thick of the wall and in the lumen of the appendix, its mesentery being oedematous and a cloudy exudate collecting in the abdominal cavity. A phlegmon of the appendix often causes infiltrations which are easily palpable through the abdominal wall. A total cessation of blood supply to the appendix due to occlusion of a vessel with a thrombus results in gangrene of the vermiform appendix.

Among the complications of acute appendicitis the most formidable is perforation or rupture of the vermiform appendix, the development of local and generalized peritonitis, and a liver abscess.

Acute appendicitis sometimes takes a chronic course. In this condition, the mucous membrane of the appendix in the ulcerated area heals or is replaced by a granulation tissue and a scar. A considerable proliferation of connective tissue occurs in the submucous and subserous layers, which ultimately leads to sclerosis and deformation of the vermiform appendix. The latter's lumen is considerably narrowed or completely obliterated. It contains a collection of serous fluid (dropsy of the vermiform appendix). The appendix may become rigidly attached or take an unusual position due to adhesions.

Clinical picture. Acute appendicitis occurs in all age groups. It is more frequent, however, in women from 20 to 40 years of age. Cases of appendicitis are known to have occurred in infancy and in very old age.

Acute appendicitis usually sets in suddenly with sharp, sometimes paroxysmal pains, which are initially periumbilical or epigastrial, and then extend to the caecum or become generalized in the abdomen. The pain is aggravated in deep inspiration and coughing, does not radiate, and is accompanied by rumbling and gurgling in the abdomen, nausea, retention of stools and gases. The temperature is normal or subfebrile, and there is a moderate leucocytosis. The ESR is initially normal and later is elevated. The pulse is quickened, but is not faster than 90-100 beats per minute; the tongue is coated and dry.

In phlegmonous-ulcerative appendicitis fever climbs up to 39-40°C, the pains become excruciating and are located in the epigastrium or the right side of the abdomen.

An attack of appendicitis sometimes presents an atypical picture. The pains may be felt in the lumbar region if the disease process is located behind the ascending colon. In elderly persons acute appendicitis may cause only slight reaction marked by insignificant pains, a normal temperature, and a low-degree leucocytosis. Some patients complain of a digestive disorder (eructation, rumbling in the abdomen, meteorism, stools of a putty consistency) and treat themselves with purgatives and enemas for constipation,which is likely to entail perforation of the appendix and peritonitis.

In diagnosing acute appendicitis great importance is attached to examination of the abdomen, which must be carried out with utmost caution. Tenderness may be discovered in the right iliac region as well as about the caecum. Less frequently a defensive rigidity of the muscles in a circumscribed area is found. Blumberg-Shchetkin's sign is the most valuable for a diagnosis; after a slight pressure applied to the abdominal wall the hand is quickly withdrawn. If an acute pain is felt by the patient at this moment, one should consider the likelihood of irritation of the peritoneum or peritonitis. Other diagnostic landmarks are Sitkovsky's and Rovsing's signs. The former consist in an aggravation of pains in the right iliac region when the patient is lying on his left side; the latter, in pains in the region of the caecum being intensified by jerky pressure applied to the descending colon below the splenic flexure.

In all cases when acute appendicitis is suspected, digital examination should be undertaken through the rectum in men and through the vagina in women; in appendicitis a site of tenderness will be felt in the lowest segment of the abdominal cavity.

An attack of acute appendicitis usually lasts 3-4 days, after which the temperature returns to normal, abdominal pains abate, only a moderate tenderness is felt in the right iliac region on palpation, the intestinal gases begin to escape and spontaneous normal stools make their appearance. Sometimes even a mild form of acute appendicitis suddenly takes a severe course and may result in perforation. In such cases the abdominal pains become generalized, Blumberg-Shchetkin's sign appears, the abdomen becomes distended, intestinal gases fail to escape, and the stool is delayed. Intestinal sounds are not audible on auscultation because of intestinal paresis. The abdominal wall becomes rigid, fever rises to 39-40°C, the systolic arterial pressure decreases and the diastolic one increases. The pulse becomes thready, and the white cell count is increased at the expense of rod neutrophils.

An infiltrate usually appears on the third or fourth day after the initial attack. Percussion of the right iliac region reveals an area of marked dullness with a circumscribed peritoneal irritation. The infiltrate is felt as a tender mass with distinct boundaries. With a progression of infiltration the temperature rises considerably and neutrophilic leucocytosis is noted. Infiltrates are usually resorbed within 4-6 weeks in response to therapy. The disease process takes an entirely different course if the infiltrate suppurates, i.e., in case of abscess formation. The patient's condition sharply deteriorates, the temperature becomes hectic, and a marked neutrophilic leucocytosis appears. The infiltrate increases in size and becomes extremely tender on palpation. Fluctuation pointing to suppuration of the infiltrate is palpable through the abdominal walls. In such a case the abscess must be urgently opened in order to avoid its rupture into the abdominal cavity and the development of peritonitis.

Acute appendicitis may become chronic, running its course with periodic exacerbations. In this condition the patient complains of discomforting sensations in the epigastrium, the right half of the abdomen, and the region of the caecum on straining, in jolty riding, climbing stairs, and fast walking. The pains radiate to the right leg or the urinary bladder. Chronic appendicitis is characterized by a permanent tenderness in the right iliac region parallel to the inguinal ligament.

The diagnosis of chronic appendicitis is aided by x-ray examination which is undertaken not earlier than 6 hours after the patient has been given the first portion of barium. Retention of barium for over 6 hours occurs in the region of Bauhin's valve. In addition, the caecum is found to be immobile because of the presence of adhesions, and either no barium is found in the appendix or its evacuation from the appendiceal lumen is delayed.

Complications. Acute appendicitis is likely to involve the following complications: (1) perforation of the appendix with the development of general peritonitis; (2) metastatic abscesses of varied localization (subdiaphragmatic abscess, paranephritis, abscesses in the small pelvis, liver, etc.); (3) thrombophlebitis of the femoral veins; (4) septicaemia.

Differential diagnosis of acute appendicitis is in certain cases very difficult because the symptom complex of acute appendicitis may be encountered in many other diseases. Biliary and renal colic, perforating duodenal ulcer, acute pancreatitis, inflammation of the uterine appendages, extrauterine pregnancy and other conditions may simulate acute appendicitis. A thorough clinical and laboratory study will throw a clear light upon the character of the disease.

Prophylaxis and treatment. The prophylaxis of acute appendicitis is largely based on a rational diet, regular gymnastics, particularly for mental workers, control of chronic constipation, elimination of helminths from the body, and correct and timely treatment of tonsillitis and other chronic infections. A patient suspected to have acute appendicitis should be immediately taken to a surgical hospital. *It is absolutely forbidden to apply heat, enema, purgatives and narcotics before the patient is seen by a physician in a hospital*, because all these remedies are likely to intensify the inflammation and cause perforation and peritonitis. At present every patient with acute appendicitis is subjected to surgery irrespective of the duration of the disease. An exception to this rule is allowed for patients with infiltrates who require medical therapy. They are enjoined to have complete bed rest, are given applications of cold and later of heat and antibiotic therapy. Surgery is nevertheless recommended soon after the resorption of the infiltrate.

Patients with chronic appendicitis also require surgery. Chronic appendicitis is prone to exacerbate during influenza, tonsillitis and other infectious diseases.

TUBERCULOSIS OF THE INTESTINE

Mesenteric lymphadenitis. A tuberculous lesion of the mesenteric lymph nodes commonly occurs in girls of 10 to 16 years of age, but is also encountered at an older age. A tuberculous infection of the mesenteric lymph nodes occurs through the intestine but most commonly extends from the intrathoracic lymph nodes. During caseous degeneration lymph nodes become coherent and form large aggregations which are liable to softening, suppuration, and formation of encapsulated abscesses. These formations may rupture into the intestinal cavity or produce an external fistula perforating the abdominal wall. Aggregations of lymph nodes may compress the abdominal organs and cause stenosis of the pylorus or the ureter with a corresponding clinical picture. The most frequent complication is extension of the inflammatory process to the peritoneum producing a circumscribed or general peritonitis.

Clinical picture. The patient complains of pains in the umbilical region and the lateral parts of the abdomen, which grow worse in walking and physical exertion and subside in a recumbent position, as well as after an escape of intestinal gases. The pains periodically become paroxysmal and are accompanied by a considerable elevation of temperature, vomiting, and sometimes by intestinal obstruction. In the periods of remission the temperature is subfebrile, often with slight chills and night sweats. Palpation reveals strictly localized tenderness in the ileocaecal region and a little above the navel at the level of the 2nd lumbar vertebra, corresponding to the location of the mesenteric nodes. Peritoneal irritation and muscular rigidity are absent.

Tuberculous lymphadenitis is attended with general intoxication, as evidenced by headaches, nervous disorders, a large loss of working capacity and anaemia. There is constipation which may alternate with diarrhoea. The stools are sometimes clay-coloured, malodorous and fatty because of malabsorption of fat due to obstruction of the lymphatic channels. In such cases the patient becomes emaciated, presenting a clinical picture known as tabes mesaraica.

The diagnosis of tuberculosis of the mesenteric lymph nodes involves great difficulties. It is made easier by the presence of a positive Pirquet's reaction and other tuberculin tests as well as an efficacious specific therapy with streptomycin, phthivazide, and PASA.

Treatment is specific, with streptomycin, phthivazide, PASA, and a cautious irradiation with ultraviolet light. Antibacterial therapy is conducted continuously during at least 6-9 months. The diet must be nutritious, rich in vitamins, and with a prevalence of proteins. Intake of vegetable cellulose and carbohydrates are limited for fear of a possible intensification of the fermentative processes in the intestine.

Tuberculous ileotyphlitis (tuberculosis of the caecum). In the opinion of most contemporary authors, this disease is associated with pulmonary tuberculosis. It is encountered both in neglected cases and in the early periods of pulmonary tuberculosis. Some authors attribute the origination of this disease to the swallowing of infected sputum. Hematogenous and lymphogenous routes of spread of tuberculous lymphadenitis also play an important role. Tuberculous ileotyphlitis is also observed in tuberculosis of the liver and the genital organs (spermatic cord, seminal vesicle, uterine appendages), and occurs by extension of the disease from the genital organs to the mesenteric lymph nodes and to the ileocaecal region via the lymphatic channels. Important factors contributing to the development of tuberculous ileotyphlitis are unhygienic habits and surroundings, mental and physical overstrain, unfavourable living and housing conditions, pregnancy, labour, abortion, and a history of gastrointestinal diseases.

Pathologic anatomy. Tuberculous ileotyphlitis occurs in the following forms: (1) ulcerative; (2) ulcerative-hypertrophic; (3) cicatricial-stenotic. The disease process is localized to the lower loop of the ileum and the caecum. In the ulcerative form the ulcers develop along the course of the ileocaecal canal, their margins are infiltrated and covered with caseous tubercles. In the hypertrophic form, the caecum is thickened and indurated due to connective tissue proliferation and infiltration of all its coats, while its lumen is narrowed.

The tubercles formed in ileotyphlitis are few in number, grow to the size of a pea, and undergo slow caseous degeneration and softening. The ulceration extends to all coats of the intestinal wall and produces circumscribed peritonitis as well as wide adhesions to the abdominal organs. As a result, perforation of a tuberculous ulcer is an extremely rare occurrence.

Clinical picture. Tuberculous ileotyphlitis is commonly encountered in women of 20 to 30 years of age but sometimes at an older age. The disease develops gradually. The initial complaints are those of indisposition, early fatigue on exertion, lassitude, headaches which are due to intoxication, nausea, vomiting, heartburn, permanent or periodic intense pains mainly localized to the right iliac region. The pains are caused by intestinal spasm, stretching of the intestinal loops and mesenteric ligaments, and sometimes by stenosis of the intestine. In the latter case, the pains become unabated and are accompanied by a loud rumbling in the abdomen. Frequently there is a periodic rise in temperature to 37.2-37.4°C. Later a constant subfebrile temperature sets in, which becomes febrile in the evening. In the period of decomposition of tuberculous tissue, formation of ulcers and peritoneal irritation there is a large difference between the morning and evening temperatures.

The tongue is usually dry and coated with a whitish film. The secretory function of the stomach is reduced to the degree of achylia. A moderate pain is felt on percussion of the right iliac region. Palpation of the abdomen reveals rigidity of the abdominal wall. In neglected cases, the indurated and thickened walls of the caecum and the terminal loop of the ileum are palpable, the latter being extremely tender on palpation.

The liver is enlarged. The heart sounds are muffled and precordial pains, extrasystolic arrhythmia, tachycardia and hypotension are encountered. In the majority of cases the lungs exhibit no active tuberculous process but shadows of calcified or fresh foci may be discovered in the roentgenogram. The peripheral blood shows a slight hypochromic anaemia which may grow to a fairly high degree if the disease process tends to linger. In periods of a relapse there is moderate neutrophilic leucocytosis with an increase in the number of rod neutrophils, lymphocytosis, and monocytosis; the ESR is permanently elevated. Tubercle bacilli are found in the faeces only in a very small number of cases. The test for occult blood is positive. The pus formed on the ulcerated surface and the inflammatory exudate are quickly carried into the distal segment of the intestine without undergoing digestion and absorption, as a result of which Triboulet's test is positive. A positive Triboulet's test and the presence of occult blood in the faeces are both important for diagnosis only in combination with other clinical, laboratory, and x-ray findings. A reliable identification of tuberculous ileotyphlitis is possible with an x-ray examination using a contrast suspension introduced by means of enema. The following x-ray findings are important for diagnosis: (1) a filling defect in the ileocaecal region; (2) absence of haustration; (3) shortening of the caecum and the ascending colon; (4) narrowing of the caecal lumen; (5) retention of barium in the caecum, while it has been eliminated from the remaining portion of the intestine. This picture is not characteristic of the initial forms of the disease.

The disease runs a chronic course. Remissions alternate with relapses which are particularly frequent in spring and autumn. Exacerbations are often attributed to dysentery, food poisoning, artificial abortion, and pregnancy. In a period of exacerbation there are new eruptions of tubercles in the ileocaecal portion of the intestine, infiltration of the intestinal walls, and caseous disintegration of the tubercles. In the period of abatement a resolution of inflammation and cicatrization of the ulcers take place.

Severe forms of tuberculous ileotyphlitis may be accompanied by serious complications. These are as follows: (1) partial or total obstruction due to a reflex spasm of the intestine, or its stenosis resulting from cicatrization of the ulcers or invagination of the ileum into the caecum. The clinical course is identical with that of intestinal obstruction of other aetiology; (2) circumscribed peritonitis due to perforation of tuberculous ulcers. A perforation is, as a rule, concealed due to the presence of adhesions between the parietal peritoneum and the serous coat of the ileocaecal portion of the intestine. Perforation is accompanied by a local rigidity of the abdominal wall, pains at the site of perforation in the right iliac region, tachycardia, and neutrophilic leucocytosis; (3) haemorrhage is a very rare complication. It is produced by ulceration of a large blood vessel.

Treatment. The disease is treated with phthivazide (0.5 g twice daily), streptomycin (500,000 U twice in 24 hours) and PASA (8-12 g daily). A prolonged (for at least 6-9 months) complex treatment is recommended particularly in the acute period. Fairly good results have been obtained by applying streptomycin in combination with thibone (0.05 g 2-3 times daily in a total dose of 8-12 g for a course of treatment).

Upon completion of the principal course of specific therapy, the latter is conducted in the form of repeated courses for a period of 2-3 years at an ambulant centre or a sanatorium.

The diet must be adequate with regard to its content of proteins, carbohydrates, fats, and vitamins. Fruit, vegetables, berries, and berry and fruit juices are recommended. For concomitant functional disturbances in other abdominal organs substitution therapy is used: for achylia, hydrochloric acid with pepsin, betacide, and acidolpepsin (tablets containing pepsin and betaine hydrochloride); for an impairment of pancreatic external secretion, pancreatin; belladonna with papaverine, platyphylline, as well as oil enemas (olive oil, sunflower seed oil given in doses of 200-300 g at bedtime) for spastic constipation and meteorism.

Tuberculous ileotyphlitis readily responds to irradiation with ultraviolet light, both artificial (quartz lamp) and natural (heliotherapy).

CANCER OF THE INTESTINE (CANCER INTESTINORUM)

Carcinoma of the duodenum (cancer duodeni) is encountered extremely rarely, mainly at ages of 40 to 60 years.

The aetiology is still unknown. There is evidence of an association existing between cholelithiasis and duodenal cancer. It is believed that in such cases the cause of carcinoma is irritation of the duodenum by passing bile calculi, the formation of ulcers followed by their cicatrization, and growth of a tumour at the site of the scar. Another factor supposed to contribute to the development of cancer is the irritating effect of secretions of the stomach, pancreas, and liver. The aetiologic role of duodenal ulcer in the development of duodenal cancer is disputed, because carcinoma is most commonly localized to the descending portion of the duodenum, to the region of Vater's ampulla, whereas an ulcer is confined to the upper portion. Duodenal carcinoma belongs to the group of adenocarcinomas, and takes its origin from Lieberkuehn's glands, infiltrates the duodenal wall, ulcerates, and causes stenosis of the intestine. Metastases occur but rarely.

Clinical picture. A carcinoma localized to the upper horizontal portion of the duodenum produces a picture of stenosis characteristic of obstructing tumours of the pylorus; and one localized to the lower horizontal portion, a picture typical of stenosis of the upper portion of the small intestine. A tumour compressing the bile ducts gives rise to mechanical jaundice. A patient with stenosis complains of vomiting stained with bile, while the gastric contents may be found to contain diastase, lipase, and trypsin. The disease runs a violent course, and does not last longer than 18 months. Anaemia and cachexia develop as a result of malabsorption in the small intestine and cancerous intoxication.

Cancer of the jejunum and ileum (cancer jejuni et ilei). Cancer of the small intestine accounts for 1-2 per cent of gastrointestinal tumours. The disease occurs more frequently in men of 40 to 60 years of age, and is predominantly localized to the ileum. The aetiology is obscure; apparently, some of the contributing factors are chronic inflammatory diseases, polyps, and injuries to the small intestine.

Cancer of the small intestine is known to occur in the polypoid and infiltrating forms. The former results in intestinal obstruction, the latter, in contraction of the small intestine. Metastases are seen in the paravertebral, supraclavicular, and cervical lymph nodes, bones, ovaries, and kidneys. For its microscopic structure cancer of the small intestine belongs to the group of adenocarcinomas, less frequently, to the group of acinous carcinomas. Cancer of the small intestine invades the large intestine, urinary bladder, and gallbladder producing pathologic communications between them. It is characterized by a rapid emaciation due to malabsorption.

Clinical picture. The initial symptoms are localized abdominal pains and constipation alternating with diarrhoea. In stenosis the upper portions of the intestine become inflated, with collapse of the lower portions and vomiting. The tumour is palpable only when it has grown to a considerable size and is rigidly held in place by adhesions. Cancer of the small intestine may be accompanied by intestinal obstruction, as well as perforation into the abdominal cavity, or the adjacent loops of the small or large intestine in consequence of ulceration or overstraining of the intestinal wall above the tumour, with formation of interintestinal fistulas. The following x-ray findings are characteristic of cancer of the small intestine: stenosis of the intestinal lumen, marginal, central, and circular filling defects, irregular contours, destruction of the mucosal folds and rigidity of the intestinal walls. The only radical treatment is surgery.

Carcinoma of the colon (cancer coli). Among malignant tumours of the gastrointestinal tract carcinoma of the colon is the fourth most frequent disease following in the order of prevalence carcinoma of the stomach, oesophagus, and rectum. In recent years, carcinoma of the colon has shown a trend toward an increase in incidence. This is probably accounted for by improved diagnostic procedures as well as by an actual rise in the rate of this disease owing to a growth in the expectation of life and an increase in the number of people living to an old age in which cancer is more frequent.

Aetiology and pathogenesis. The aetiology of carcinoma of the colon is unknown. An important role in its genesis is apparently played by polyposis, inflammatory diseases, and mechanical damage. Evidence in favour of the latter is frequent localization of carcinoma to the sites of the physiological narrowing of the colon and congestion of faecal matter in the hepatic and splenic flexures, the caecum, and the sigmoid colon. Other important factors contributing to the genesis of cancer of the colon are constipation, chronic colitis, bacterial and amoebic dysentery, and chronic ulcerative colitis of unrecognized aetiology. For their microscopic structure 95 per cent of carcinomas of the colon belong to the group of adenocarcinomas, although acinous and mucous carcinomas are also encountered.

Clinical picture. Carcinoma of the colon usually occurs in persons of 40 to 60 years of age, and is equally frequent in both sexes. The initial symptoms are highly uncertain and nonspecific. In some patients the disease sets in with dyspeptic symptoms (eructation, nausea, and abdominal pains), in others, a tumour is discovered by palpation in the right or left side of the abdomen. Pain is an important initial symptom of carcinoma of the colon. It is caused by intestinal obstruction, ulceration of the tumour, and irritation of the peritoneum. A number of symptoms vary with the localization of the malignant neoplasm. When it is localized to the left colon where faecal matter is formed, signs of intestinal obstruction and pains appear, whereas a tumour growing in the right colon, the contents of which are liquid, causes malabsorption. A left-side carcinoma produces a local distention, a visible intensified peristalsis, and a loud rumbling sound. Percussion over a tumour may elicit a dull tympanitic sound. Carcinoma of the ascending and descending colon is readily palpable. A tumour causing contraction of the intestine is palpable quite rarely. A tumour of the hepatic flexure is palpable from the front and rear. A tumour of the sigmoid flexure is sometimes palpable in a patient lying on his right side. The absence of a palpable tumour is no reason to rule out carcinoma of the colon.

A certain role in the diagnosis of cancer of the colon is played by a positive test for occult blood in the faeces, as well as the presence in faecal matter of particles of a disintegrating tumour. The principal diagnostic procedure is x-ray examination with introduction of a contrast substance through the mouth or by means of enema. In the presence of tumours localized to the right colon the contrast substance is administered per os, whereas in case of a suspected tumour in the left colon a barium enema is prescribed. The x-ray picture of carcinoma of the colon presents the shadow of a tumour seen as a filling defect with irregular, as though nibbled, margins. The intestinal lumen is stenosed and fails to distend when filled with barium.

Course and complications. Carcinoma of the colon progresses very slowly, and is attended with growing debility, hypochromic anaemia and emaciation, while metastases develop later than those from carcinoma of another localization. Among the complications the most formidable is intestinal obstruction caused by a gradual narrowing of the intestinal lumen to the degree of its complete occlusion by the tumour. There is constipation, an intensified local peristalsis with intestinal distention, rumbling, and persistent vomiting. With a tumour localized to the right colon, obstruction occurs rarely, which is probably due to the wide lumen of the intestine and its liquid contents. Cancer of the colon may become the cause of invagination, sometimes over a considerable length. In a few cases, there is perforation and rupture of the intestinal wall followed by peritonitis. Of the other complications of carcinoma of the colon mention should be made of its extension to the adjacent organs—the gallbladder and the urinary bladder from the sigmoid colon, and to the adjacent parts of the large and small intestine from the caecum, as well as an adhesive inflammation between the intestinal loops with the development of general peritonitis.

Treatment is exclusively surgical. An early diagnosis of carcinoma of the colon and, consequently, timely surgical intervention give very favourable immediate and remote results. Surgery must necessarily be followed by radiotherapy. For multiple metastases radiotherapy must be supplemented with antitumour chemotherapy, with sarcolysin in particular. The latter is administered intravenously with a 15-20 per cent glucose solution or a physiologic salt solution in doses of 0.2-0.3 g gradually increased to 0.6 g in 24 hours. The total dose of sarcolysin for one course of treatment is 6 g. Both radiotherapy and chemotherapy are carried out with a check kept over the morphologic composition of the white blood.

Carcinoma of the rectum (cancer recti). In respect of frequency carcinoma of the rectum ranks third among malignancies of the stomach and intestine after carcinoma of the stomach and oesophagus.

The *aetiology* of this disease is unknown. Only the existence of certain predisposing factors has been supposed. Among them are congestion of faecal matter, ulceration of the rectal mucosa of varied origin, cicatricial-ulcerative changes and strictures of the rectum, haemorrhoids, and malignant degeneration of polyps. Cancer rarely extends all over the length of the rectum, and is more often confined to the ampulla, less frequently to the anus. For their histologic structure rectal cancer cells are cylindrical, acinous and mucous. The tumour invades the perineum and the organs of the small pelvis. Metastases are usually found in the lymph nodes, liver, lungs, spinal column, urinary bladder, bones, etc.

Clinical picture. The disease is relatively slow in development. Pain is one of the early signs of rectal carcinoma, but it may also appear at a later time as a result of extension of the tumour to the sacrum and coccyx. Rectal cancer runs its course with intermittent constipation and diarrhoea due to the development of an inflammatory process. With a tumour localized to the ampulla of the rectum the faeces are blood-streaked, and blood-stained mucus discharge is notable. Blood discharged through the anus may be dark because of blood retention in the rectal ampulla. Constriction of the anus and formation of fistulas discharging faecal matter, pus and mucus may also occur. In the late period of the disease there is stubborn constipation due to a mechanical obstruction. In case of persistent constipation developed at an advanced age careful examination for rectal cancer is necessary. Partial or total obstruction due to occlusion of the rectum by a tumour may be found. Rectal carcinoma gives rise to serious complications in the adjacent organs; extension of the tumour to the urinary passages with dysuria, a vesiculorectal cloaca with a simultaneous discharge of faecal matter and urine through the rectum. A rectovaginal fistula may develop in women. Metastases in the bones cause pathologic fractures.

Rectal carcinoma is diagnosed by means of digital and x-ray examination, rectoromanoscopy and biopsy. Digital examination may help determine the presence of a tumour, the degree of its mobility, its form, consistency, relation to the surrounding organs, as well as metastatic nodes in the thick of the intestinal wall. Rectoromanoscopy makes it possible to discover a tumour, perform a biopsy, and take smears for a subsequent histologic study. X-ray examination is important, above all, in the presence of rectal carcinoma of a high localization. Cancer of the rectum is characterized by the following x-ray findings: discontinuity of the mucosal folds, a parietal stenosing defect, a filling defect, a niche in the defect (in ulceration), rigidity of the intestinal wall, stenosis of the lumen and its distention above the site of constriction.

The *treatment* is surgical. A radical operation is the most efficacious if cancer has not extended beyond the rectum.

HELMINTHIASIS

The following three classes of helminths are the most widespread: (1) threadworms (*Nematoda*)—ascarides, pinworms, whipworms (*Trichuris trichiura*), hookworms (*Ancylostoma*), *Trichinella* parasites (trichinae), etc.; (2) tapeworms (*Cestoda*)—*Bothriocephalus*, *Taenia saginata* (beef-tapeworm), *Taenia solium* (pork tapeworm), dwarf tapeworm, *Taenia echinococcus*, etc; (3) *Trematoda* worms (flukes)—cat fluke, liver fluke, lanceolated fluke, etc.

Nematodiasis. The *Nematoda* helminths are the most widespread. Almost all of them are heterosexual, males being smaller than females and having spiral-shaped tail-ends as distinct from the latter. They have a digestive tube and mouth parts surrounded by growths and "lips".

Ascariasis (ascaridosis) is a highly prevalent disease due to infestation with ascaris (*Ascaris lumbricoides*). The ascaris is of a reddish colour, resembling an earthworm, and usually inhabits the small intestine. A female worm daily lays about 200,000 fertile eggs in the host intestine. In case only males are present in the intestine, no eggs are found in the faeces, whereas in the opposite case, i.e., in the presence exclusively of females, the faeces contain only unfertile eggs of an elongated pear-like form.

After discharge from the intestine, a fertile ascaris egg develops to the larval stage under proper conditions of moisture, aeration, and temperature ranging from +24 to —30°C. At a low temperature (but not below —30°) the eggs remain viable but their development is retarded. At least 10 days are required for an egg to develop into a larva. Therefore, newly-discharged faeces are not infective (Fig. 61).

Spring and autumn months and a humid climate are especially favourable for the development of larvae. In districts with a dry climate the incidence of ascariasis is lower because ascaris eggs (ova) perish on exposure to sunlight in summer, particularly when deposited in sand.

Ascariasis is more frequent where sanitary and hygienic control is inadequate. In man the disease follows the ingestion of embryonat-

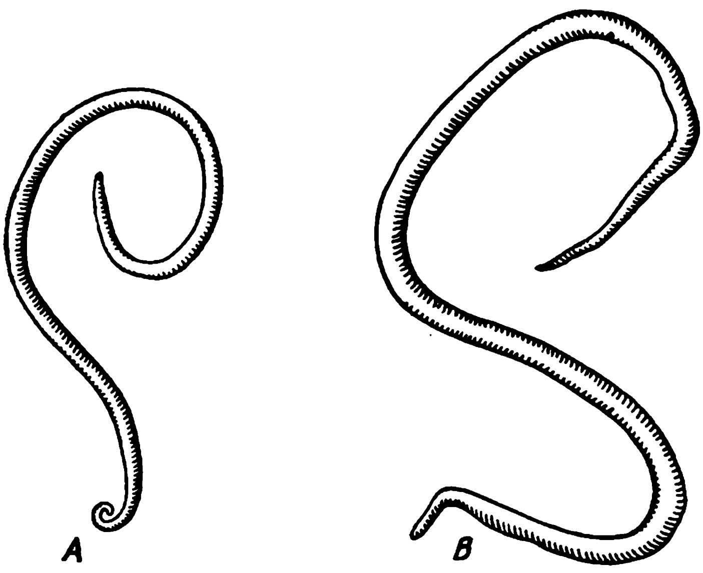


Fig. 61. Ascarides
A—male; *B*—female

ed eggs of ascarides, usually with food (unwashed raw vegetables, fruit, berries) and water. In the intestine the eggs hatch and the cycle of larval development begins. A newly-hatched larva penetrates the mucous membrane of the small intestine, migrates through the system of the portal vein into the liver, from the latter through the inferior vena cava to the right heart, and from there to the lungs. The larva is detained in the pulmonary capillaries, then bores the capillary wall to pass into the alveoli, smaller bronchi and trachea. Further, the larva enters the mouth with the sputum during coughing and is swallowed. On re-entry into the intestine it can develop into an adult parasite.The entire life cycle, from the larval stage to the adult, lasts 6 to 8 weeks.

Clinical picture. The patient complains of abdominal distention and epigastric pains appearing after a meal; sometimes umbilical pains, anorexia, and aversion to certain foods are observed. Sometimes there is a loose stool and salivation. Nervous disorders are manifested by an early fatigue on exertion, irritability, headaches, dizziness, twitchings, usually at night, and night terrors. Often the patient grits his teeth in sleep. Sensitization of the body with the products of helminth metabolism absorbed into the blood may result in spastic contractions of the bronchi to the degree of typical attacks of bronchial asthma, urticarial rashes (nettle-rash, urticaria), etc. Anaemia is relatively infrequent, and the number of eosinophils is often elevated.

The following complications of ascariasis deserve attention: (a) obstruction (or occlusion) of the physiologically narrow parts of the intestine; (b) perforation of the intestine; (c) lobular pneumonia. Obstruction of the intestine by an aggregation of ascarides or by one parasite, in the presence of an intense intestinal spasm, may interfere with the passage of chyme. This gives rise to pains which resemble in character those felt in cholelithiasis or nephrolithiasis. Migration of ascarides into the liver and bile ducts may be accompanied by cholangitis, cholecystitis, abscess of the liver, and mechanical jaundice. Ascarides entering the vermiform appendix may cause appendicitis.

The diagnosis is made by detecting ascarides or their eggs in the faeces. In the presence of ascarides and absence of eggs in the faeces there is reason to suspect natural decrepitude or immaturity of females as well as an infection by males only. Helminths in the intestine are detected by x-rays in the form of worm-shaped pale shadows seen against the background of an intense shadow of the intestine.

Treatment and prophylaxis. The best prophylactic measure is observance of the rules of personal hygiene. Other important prophylactic steps are as follows: (a) construction of public lavatories designed to prevent contamination of soil and water with eggs of helminths; (b) decontamination of refuse, fertilizers and sewage; (c) implementation of mass-scale anthelmintic treatment, particularly among inhabitants of hostels and in large collective bodies. It is necessary to explain the dangers of eating unwashed raw vegetables and to destroy flies, cockroaches, and rats which are likely to transmit ascaris eggs.

Ascariasis is treated with piperazine, santonin, oil of chenopodium, and oxygen. Piperazine-adipinate is administered in 1 g doses three times daily an hour after a meal for two days running. Pretreatment purgation is not prescribed. For constipation purgatives (purgen [phenolphthalein], rhubarb) are administered in the evening on the second day of treatment. No contraindications for piperazine are known. Santonin apparently fails to kill ascarides but only stuns and drives them into the large intestine from which they are expelled with saline purgatives. Two days before, during and after santonin treatment the patient is given semiliquid, mainly carbohydrate and lean food. On the evening preceding treatment he is given a purgative in order to cleanse the intestine and increase the exposure of the parasites to santonin. This is especially important in case of constipation. There are two methods of santonin therapy. By the first method the preparation is prescribed in doses of 0.08-0.1 g to be taken three times daily $1^1/_2$-2 hours before a meal. On the second day of treatment, after taking santonin, a saline purgative is administered.

By the second method, treatment is continued during 3 days. Santonin is taken in the morning on an empty stomach in doses of 0.1 g three times at one hour intervals. Within an hour of taking the final dose of the preparation a saline purgative is administered, and a light breakfast is given after another hour. Treatment by the latter method is poorly tolerated by children under 3 years of age and debilitated adults. Santonin treatment may be repeated only in 2-3 weeks' time. Wormseeds (dried flower heads of santonica) are sometimes used instead of santonin in a dose of 5 g, ground with sugar, honey or jam, to be taken three times daily during a period of two days. The day before and during wormseed treatment purgatives are administered. Oil of chenopodium (Ol. Chenopodii) is also used in the treatment of ascariasis. It is contraindicated for infants, elderly persons, and in diseases of the liver, kidneys, heart, gastrointestinal tract, and in pregnancy. Oil of chenopodium is administered in gelatin capsules (in view of its irritating effect on the gastrointestinal

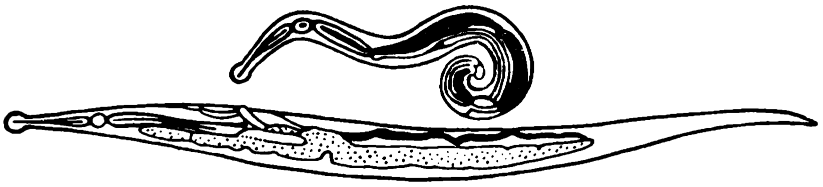


Fig. 62. Pinworm

mucosa), or on a piece of sugar. It is given on an empty stomach or 3 hours after a breakfast in a dose of 1 ml three times at one hour intervals. No pretreatment purgation is necessary. An hour after taking the final dose, a saline purgative, Vienna drink (senna), or purgen is administered. Oil of chenopodium may be administered in combination with castor oil (Ol. Chenopodii 20.0, Ol. Ricini 50.0).

Ascariasis is also treated by introducing oxygen into the stomach, either on an empty stomach or 2-3 hours after a meal, in a dose of 1.5 litres per injection for two days running. Oxygen is injected slowly through a thin gastric tube in doses of 200-250 ml at 1-2 minutes intervals by means of an apparatus for applying artificial pneumothorax or Bobrov's apparatus. The entire procedure lasts 15 minutes. Upon completion of injection the tube is withdrawn and the patient rests in a recumbent position for two hours, after which he is allowed a meal. A saline purgative is prescribed for constipation on the next day after treatment. Oxygen therapy is contraindicated in diseases of the gastrointestinal tract (gastric and duodenal ulcer, tumours, etc.).

Control examinations of the faeces must be carried out 2-4 weeks after the completion of therapy. For prophylactic purposes a mass-scale piperazine or santonin treatment of large collective bodies is recommended in spring and autumn when infection with ascariasis usually takes place. The life of an ascaris lasts for 18 months, therefore reinfection may be prevented by vigorous prophylactic measures.

Enterobiasis (enterobiosis). This disease in due to infestation of man by pinworms or seatworms (*Enterobius vermicularis*) and is one of the most common helminthic infections (Fig. 62). It is more frequent in children of school and preschool age. The eggs of pinworms are elongated in form, have a smooth doubly contoured clear membrane with larvae in its centre. Pinworms (usually numbering several thousands) invade the upper portion of the large intestine—the caecum, the vermiform appendix, and the lower portion of the small intestine. Here they attach themselves by suction to the mucous membrane and sometimes penetrate rather deeply into Lieberkuehn's glands. The uterus of a gravid female contains about 1,200 eggs. Males die after copulation, while females migrate to the lower parts of the large intestine, the rectum, and, having passed the latter, deposit a large number of eggs in the skin folds about the anus and die. When the skin about the anus is being scratched, eggs may get onto the fingers and under the nails from where they may find their way to food, enter the body by mouth and cause a reinfection.

Infection may also be spread through faecal matter containing the eggs of pinworms. The eggs deposited in the perianal region are not full-grown. They reach maturity within 6-7 hours at a temperature of 36-37°C. At a temperature below 22°C this maturation fails to occur; at room temperature, however, mature eggs remain viable for a month. They may be discovered in dust on the floor, walls, window-sills, wardrobes, classrooms, etc.

Clinical picture. Penetrating into the depth of the intestinal mucosa, pinworms may contribute to infection of tissues by intestinal bacterial flora causing inflammation and provoking the development of catarrhal and purulent appendicitis. Pinworms may occasion the formation of itching anal fissures which are likely to be scratched and become suppurated. Sometimes irritative skin rashes and eczema due to irritation may be encountered. Prolonged and intense itching in the perineum may be responsible for the patient, especially a child, becoming nervous, capricious, with a loss of appetite, broken sleep and nocturnal enuresis. An intense perineal pruritis in women is accompanied by psychasthenia and a reduction in working capacity. In girls irritation of the genitalia by pinworms leads to an early sexual desire.

The diagnosis of enterobiasis is based on the patient's complaints, evidence of pinworms discharged through the anus, and detection of the parasites or their eggs in the faeces or about the anus. For this purpose, a scraping is obtained from the skin about the anus with a spatula and moistened with a 2 per cent soda solution, transferred in a drop of the same solution onto a slide, and examined under a microscope. Eggs of pinworms may also be discovered in scrapings from under the nails. The edge of the nail and the underlying skin are painted with a 1 per cent alkaline solution and wiped with a small cotton swab. Then the latter is placed in a test tube and centrifuged in a solution of caustic alkali for 3 minutes, the sediment being examined under a microscope.

Treatment and prophylaxis. For prophylactic purposes one should wash his hands thoroughly with brush and soap after a toilet, on rising from bed in the morning, and before a meal. Rooms must be periodically cleaned with a wet rag to prevent dissemination of eggs and their inhalation with dust. It is necessary to carry out a mass examination of large collective bodies for enterobiasis at least twice annually and a mass-scale anthelminthic treatment whenever this disease has been found.

The patient must wash his perianal region with warm water and soap on rising from and before going to bed for a period of four to six weeks in order to remove the eggs of pinworms, as well as paint this region with a thick layer of petrolatum to prevent pinworms from spreading. After washing himself for the night the patient must put

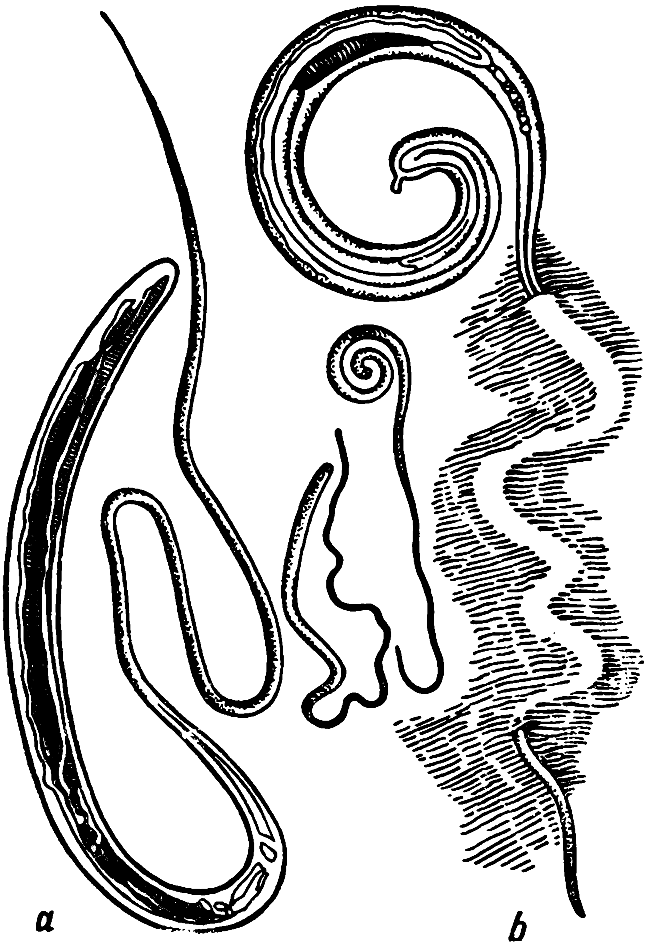


Fig. 63. Whipworm
a—male; b—female

on clean underclothes which should be taken off and boiled in the morning to destroy pinworms which have gathered on them during the night. He must sleep in close-fitting trunks, and his bedding and underwear must be ironed every day.

A helpful remedy for enterobiasis is an enema of 5 glassfuls of clear water or water with vinegar or common salt, a weak soda solution (half-a-teaspoonful of soda to each glass of water). Perianal itching may be soothed with cold lotions as well as an embrocation of mercuric sulphate ointment or a 5 per cent anaesthesin ointment (Anaesthesini 1.0, Ol. Vaselini 20.0).

The most efficacious methods of therapy are administrations of piperazine preparations (piperazine-adipinate, piperazine-sulphate, etc.), in doses of 2-3 g in 24 hours for a period of 5 days. Whenever necessary, 2-3 courses of treatment are carried out at intervals of one week. Also helpful is treatment with aspidium extract which contributes to elimination from the body of both adult parasites and larvae. It is administered to adults in a dose of 5 g twice at intervals of 10-12 days. On the evening before treatment a saline purgative is given. Aspidium is taken by the patient on an empty stomach in capsules, with semolina or with jam during 15 minutes. An hour later he is given a saline purgative and after two hours, a breakfast.

Gentian violet is administered to adults in doses of 0.06-0.08 g three times daily for a period of 5-7 days. A purgative is prescribed on the third and last days of treatment. After a 7-day interval this treatment is repeated. It is contraindicated for ascariasis and diseases of the kidneys and heart.

Trichuriasis (trichocephalosis). This disease is caused by whipworms (*Trichuris trichiura*) which parasitize the large intestine, mainly the caecum and the vermiform appendix, and may also invade the lower portion of the small intestine (Fig. 63). The eggs of whipworms maturate only in the external environment after being deposited there with the faeces. At least three weeks are necessary for them to reach maturity. The eggs gain entry to the human body with unwashed raw fruit and vegetables. They may also be spread by flies. In the human intestine the eggs develop into larvae which gradually grow into adult whipworms. By its thin head end an adult whipworm attaches itself to the mucous membrane, causing multiple lesions, sometimes haemorrhages, and infection of the intestinal wall. The parasite may live in the intestine as long as several years.

Clinical picture. Infection by isolated whipworms may be asymptomatic. A patient with a massive infection complains of headache, dizziness and epigastric pains. Whipworms are often found in the vermiform appendix, in which case paroxysmal pains like those in appendicitis occur. There may be vomiting, salivation, diarrhoea, sometimes with admixed mucus and blood, and constipation may develop as a result of intestinal spasms. In more severe cases, anaemia as well as leucocytosis with an increased number of lymphocytes and pronounced eosinophilia develop. The patient is irritable, debilitated, with a loss of weight, decreased appetite and reduced gastric secretion. In the most severe cases, epileptiform fits and sometimes fainting may take place. The diagnosis is verified by detecting the eggs of whipworms in the faeces. It is preferable to examine the faeces after they have been allowed to stand for 14-16 hours.

Treatment and prophylaxis. The remedies most commonly used for the expulsion of whipworms are osarsol (acetarsone) with methylene blue, thymol, and introduction of oxygen into the rectum. An enema of a water-benzine mixture is also recommended. Osarsol is prescribed in 0.25 g tablets to be taken one hour before meals four times daily for 5 days running. A purgative is administered on the sixth day. In order to intensify the effect of osarsol, methylene blue is given in a dose of 0.1 g 3 times daily during a period of three days before taking the drug. During therapy piquant, salted, sour and roasted foods as well as alcohol are withdrawn from the diet. Osarsol is contraindicated for diseases of the liver and kidneys, gastric and duodenal ulcer, etc. Upon the appearance of signs of poisoning (abdominal pain, vomiting, diarrhoea, headache, fever, etc.) osarsol treatment must be immediately discontinued, a purgative administered, the stomach lavaged, and 20 ml of a 10 per cent sodium hyposulphite injected intravenously.

The water-benzine mixture consists of 15 ml aviation petrol and 1 litre of water per injection. This mixture is injected into the rectum after a cleansing enema for 5 days running.

Oxygen therapy is preceded by a cleansing enema, after which 1-1.5 litres of oxygen is introduced into the rectum. The course of treatment lasts 7 days after which a saline purgative is administered for 2 days.

Prophylaxis of trichuriasis is identical with that of ascariasis.

Ancylostomiasis or hookworm disease (ancylostomidosis). The term ancylostomiasis is used to denote two helminthic diseases, one due to *Ancylostoma duodenale.* (Gr. *ancylos*—hook, *stoma*—mouth) and the other due to ***Necator americanus.*** Both helminths are parasitic in the duodenum but may also inhabit other parts of the intestine. The clinical manifestations of these helminthic infections are identical. They are widespread in tropical and subtropical countries.

Man usually acquires hookworm infection through unprotected skin, mainly of the hands and feet, when working on tea plantations, in mines, and tunnels (hence the term "tunnel anaemia" under which this disease is known also). Penetrating the skin, hookworms gain entry to the blood vessels through which they are carried to the lungs. From there they migrate into the respiratory passages through the capillaries and alveoli and are swallowed. In the intestines the parasites attach themselves to the wall. Adult hookworms damage the intact mucosa of the small intestine with the cutting plates of their mouth cavity. Subsequently the injured parts bleed continuously. Infection may also be acquired through food and water. In this case, the larvae bypass the above-described route and are detained in the intestine. The migration of larvae takes from 7 to 10 days while the entire life cycle lasts from four to six weeks. Hookworms have been known to survive in the human body for several years.

Clinical picture. The characteristic symptoms of ancylostomiasis are gastrointestinal disturbances, anaemia and nervous disorders. Urticaria, itching eruptions, lymphangitis and pyoderma may develop at the site of larval penetration. The initial symptoms are pains in the upper abdomen, debility, dizziness, dyspnoea, nausea and loss of appetite. These may later be followed by nosebleed, cardiovascular disorders, facial oedema, anaemia and a perversion of appetite (desire to eat clay, chalk, earth). The nails acquire a spoon-shaped form (koilonychia). In epidemic foci, the diagnosis is established from the presence in the patient of iron deficiency anaemia, with eosinophilia concurrent with a perverted appetite and facial oedema. The disease runs a protracted course. Uncomplicated cases end in recovery.

Treatment. The best remedy against hookworm disease is carbon tetrachloride which is prescribed in a dose of 3 ml to be taken 2-3 hours after a breakfast rich in carbohydrates. The drug is taken once in gelatin capsules or with milk (50 ml of milk). During therapy abstinence from intakes of fat and alcohol is mandatory. A quarter-hour after the administration of the drug a saline purgative is taken, and after another two hours the patient is allowed a meal. A repeated course of treatment should not be undertaken earlier than in 2 weeks' time. Carbon tetrachloride is contraindicated for diseases of the liver, kidneys, and gastrointestinal tract. Sometimes hookworm disease is treated with thymol, oil of chenopodium, hexylresorcinol, often in combination with carbon tetrachloride. Naphtammon is also used. Iron preparations and blood transfusions are also helpful.

Prophylaxis of hookworm disease consists in observance of hygienic rules and protection of exposed parts of the body against larvae when working on plantations, in mines and tunnels. All persons afflicted with ancylostomiasis must be treated.

Strongyloidiasis (strongyloidosis). This disease is mainly prevalent in tropical and subtropical countries, although sporadic cases have been reported in temperate regions also. The causal agent of this disease is a very small nematode (*Strongyloides stercoralis*). Its development is characterized by the alternation of parasitic and free-living generations. Adult females of a parasitic generation inhabit mainly the Lieberkuehn's glands of the duodenum, sometimes migrate to the mucous membrane of the pyloric part of the stomach, the entire small intestine, the caecum and colon, and even invade the biliary and pancreatic ducts. Gravid females lay eggs which develop into rhabditiform larvae discharged from the intestine with the faeces. In an unsuitable external environment the larvae develop into the filariform larval stage, and under favourable external circumstances into free-living males and females. After copulation gravid females lay eggs which develop into rhabditiform larvae. The latter may develop into another generation of free-living parasites or transform into the filariform larvae capable of entering the human body through the skin or mouth with ingested food and drink. In either case they pass through the lungs and pharynx, are swallowed into the stomach and enter the intestine, reaching the adult stage during this migration. Females may be fecundated both in the lungs and in the intestinal lumen; after copulation the males die.

Clinical picture. The patient complains of nausea, dizziness and bloody diarrhoea resulting from ulcerations due to parasites lodged within the intestinal mucosa and glands. At the onset of the disease there is itching, urticaria, papular eruptions, and local oedema at the site of larval penetration as well as bronchitis and pneumonia. Strongyloidiasis may run a mild or severe course. Mild forms are accompanied by abdominal pains, particularly in the epigastrium, nausea, salivation, alternation of constipation with diarrhoea, dizziness, irritability and eosinophilic leucocytosis (up to 40-80 per cent). In extremely severe cases, usually in the tropics, the disease may take a fatal course due to an extensive intestinal involvement.

The *diagnosis* of strongyloidiasis is made by finding highly mobile larvae in newly-voided faeces and in the duodenal contents. Diagnosis is also aided by an allergic skin test and the presence of eosinophilia at the onset of the disease.

Treatment and prophylaxis. The most efficacious remedy is gentian violet. The drug is administered in gelatin capsules in doses of 0.05-0.06 g three times daily for 8-16 days. Treatment with gentian violet sometimes causes the appearance of a pale lilac colour of the mucous membranes, slight nausea, less frequently vomiting, and abdominal pains. Prophylaxis of this disease is identical with that of ancylostomiasis.

Trichinosis (trichinellosis). This disease is a widely distributed helminthic infection and is especially common in America. It is caused by the intestinal nematode *Trichinella spiralis*. In the adult stage the parasite inhabits the small, less often the large intestine, and in the larval stage invades the striated muscles of man and other mammals. Man contracts the infection following ingestion of raw, newly-salted, slightly salted and undercooked pork, meat of wild boar, bear, and badger infected with encysted *Trichinella spiralis* larvae. During digestion of meat in the gastrointestinal tract the cysts are dissolved and the liberated larvae develop to maturity in 30-40 hours. In 48 hours the females are fecundated by males which die after copulation. From 4 to 6 days later the females discharge larvae which enter the lymphatic system and blood and settle in the striated muscles where they acquire a spiral shape in three weeks' time. Within 35-40 days of infection of man the larvae are invested in a lemon-shaped cyst which in 6 months becomes impregnated with lime salts. In the intestine *Trichinella* parasites live from 10 to 84 days and rarely longer.

Clinical picture. The varied clinical manifestations of this disease are associated with allergic reactions of the body to the effects produced by the metabolic and disintegration products of adult *Trichinella* nematodes as well as their migrating larvae. The incubation period lasts 10 to 15 days, and rarely longer. The period of precursory symptoms manifested by lassitude, dejection, headaches and sometimes gastrointestinal discomfort does not last longer than 2-3 days, and in rare cases continues for 10-20 days. The early symptoms of trichinosis are oedema of the eyelids and face, sometimes of the extremities, fever, muscular pains and eosinophilia. Oedema persists for one or two weeks. The fever is remittent or continued. Muscular pains are felt after the appearance of oedema and are localized to the muscles of the eyeball, the extremities and trunk. The muscles become indurated, and contractures of the knee and elbow joints are frequent. An abatement of pains is preceded by a fall of temperature. At the end of the first week of the disease the patient develops eosinophilia (up to 85 per cent), leucocytosis and an elevated ESR. A reduction in the number of eosinophils occurs within 2-3 months of the onset of the disease. From the seventh-eighth day, the skin of some patients exhibits maculopapular eruptions and urticaria which may disappear shortly.

Gastrointestinal disorders are found in approximately 50 per cent of patients. Trichinosis produces a number of neurologic disturbances. The patient is troubled by stubborn headaches, insomnia, debility, disappearance of tendon reflexes, and paresis and paralysis of the lateral muscles of the eye. The findings in the organ of vision are conjunctivitis, conjunctival haemorrhages and exophthalmos. In atypical forms of the disease the early signs of trichinosis are meningitis, encephalitis and hemiplegia. The duration of the disease is 2 days to 5-8 weeks. Sometimes, it runs its course with one to three relapses occurring at intervals of 2-25 days.

Among the complications of trichinosis attention is deserved by pneumonia developing in the third-fourth week of the disease, pleurisy, pulmonary suppurations, encephalitis, meningitis, stomatitis, cystitis, etc. Death results from a circulatory failure in the fourth-eighth week.

The *diagnosis* is made on the basis of symptoms characteristic of trichinosis: oedema of the eyelids, muscular pains and eosinophilia. It is aided by biopsy of the muscles and their examination for the presence of *Trichinella* parasites. Identification of the disease is also facilitated by intracutaneous and serologic immunologic tests, which may be positive only in the second or third week of the disease. The diagnosis is also made easier by the discovery of larvae in remains of ingested meat. From the sixth month of the disease the diagnosis can be aided by x-rays which reveal calcified *Trichinella* cysts in the muscles.

Treatment and prophylaxis. All patients with trichinosis require hospitalization. No specific remedies against this disease are available, therefore symptomatic therapy is given. In view of the allergic pathogenesis of trichinosis, ACTH, cortisone, prednisolone, etc., are used effectively. ACTH is administered intramuscularly in a dose of 15 U once in every six hours, and prednisolone and other similar preparations, in doses of 25-30 mg 2-3 times daily for a period of 2-3 weeks. During this treatment the patient is maintained on a diet rich in potassium salts (potatoes, raisins, dried apricots, etc.) and poor in chlorides. In the case of a relapse steroid hormone therapy is resumed. The diet must be nutritious, with a liberal liquid intake. Dehydration of the body is controlled by subcutaneous infusions of a physiologic salt solution and glucose, blood and blood plasma transfusions, vitamins, etc. In the presence of muscular pains active movements must be restricted. For myocarditis cardiac drugs and diuretics are prescribed, for pneumonia, antibiotics and sulpha drugs. To accelerate calcification of the cysts calcium chloride, a diet rich in calcium salts, and vigantol (vitamin D) are given.

Prophylaxis of trichinosis consists in a veterinary and sanitary inspection of slaughter livestock. Under Soviet legislation all *Trichinella*-infested carcasses are subject to technical utilization. In trichinosis control great importance is attached to destruction of rats and mice, a rational management of pigsties, trichinelloscopy of all slaughtered pigs, a thorough processing of tinned pork, and precautions against eating lard. A substantial role is played by epidemiologic surveys undertaken immediately after detection of a new case of trichinosis.

Cestodiasis or tapeworm infections. *Diphyllobothriasis.* This disease is caused by the broad tapeworm or fish tapeworm (*Diphyllobothrium latum*) which is parasitic in the small intestine of man, the dog, the cat and the hog. It enters the intestine together with undercooked fish (Fig. 64).

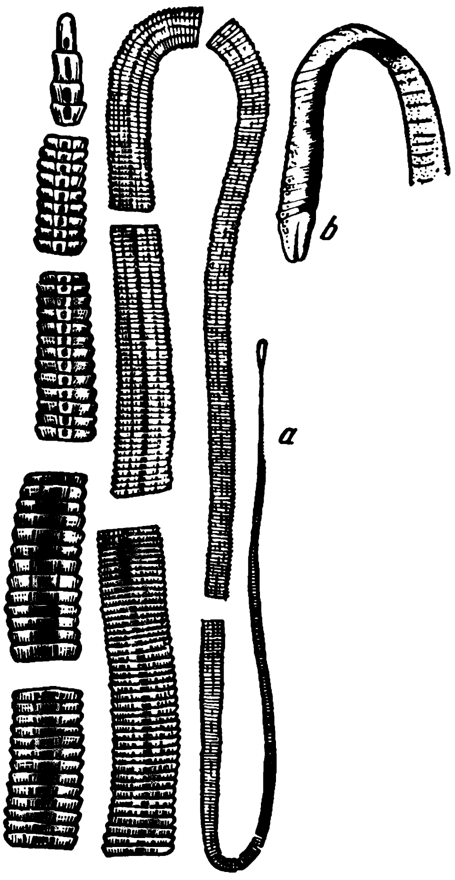


Fig. 64. Broad tapeworm
a—life-size picture of scolex; *b*—enlarged picture of scolex

The adult worm has the shape of a tape measuring from 2 to 10 m in length. It consists of the head, neck and body. The head has two suckers or bothridia (Gr. *bothria*—pits) by which the parasite attaches itself to the intestinal mucosa. The neck is thread-shaped, while the body consists of segments, the so-called proglottids, the width of which is larger than the length. In the central part of each segment is found a rosette-shaped loopy brown-coloured uterus loaded with large eggs. Through an opening at one of the ends of the uterus the eggs are discharged into the intestine. The eggs are oval in shape and are enclosed in a smooth grey-coloured membrane. They are eliminated with the faeces and develop further only if they get access to water where they mature to the larval stage known as coracidium on which cyclops feed. In the cyclop body the coracidium develops to the second larval stage known as the procercoid. Cyclops are in their turn eaten by perch, burbot, salmon and trout. Within the fish body the procercoids develop to the plerocercoid stage which may migrate from one fish to another when its host is ingested by rapacious fish. Man acquires diphyllobothriasis following ingestion of raw or undercooked infected fish. In the human body the plerocercoid develops into an adult parasite in 4-6 weeks.

Clinical picture. There is loss of appetite, nausea, diarrhoea, lassitude, salivation, palpitation, dizziness, dyspnoea, and a discharge of scraps of tapeworm proglottids, the latter sometimes being the patient's only complaint. The broad tapeworm absorbs a very large amount of vitamin B_{12}, therefore, the disease is sometimes complicated by a condition resembling pernicious anaemia with a high colour index and marked eosinophilia. In such cases there is pallor of the face and skin, a painful tongue (stomatitis), tenderness of the sternum on slight strokes, enlargement of the spleen and liver, achylia and neurologic disorders. Upon expulsion of helminths all these symptoms, including achylia, disappear.

The diagnosis is made by the finding of proglottids or eggs in the faeces.

Taenia saginata infestation occurs in persons who have the custom to eat raw or undercooked beef.

The causal agent is the beef tapeworm (Fig. 65) which measures 4 to 10 m in length. Segments of this worm may crawl out through the anus independently. In this case and when discharged with the

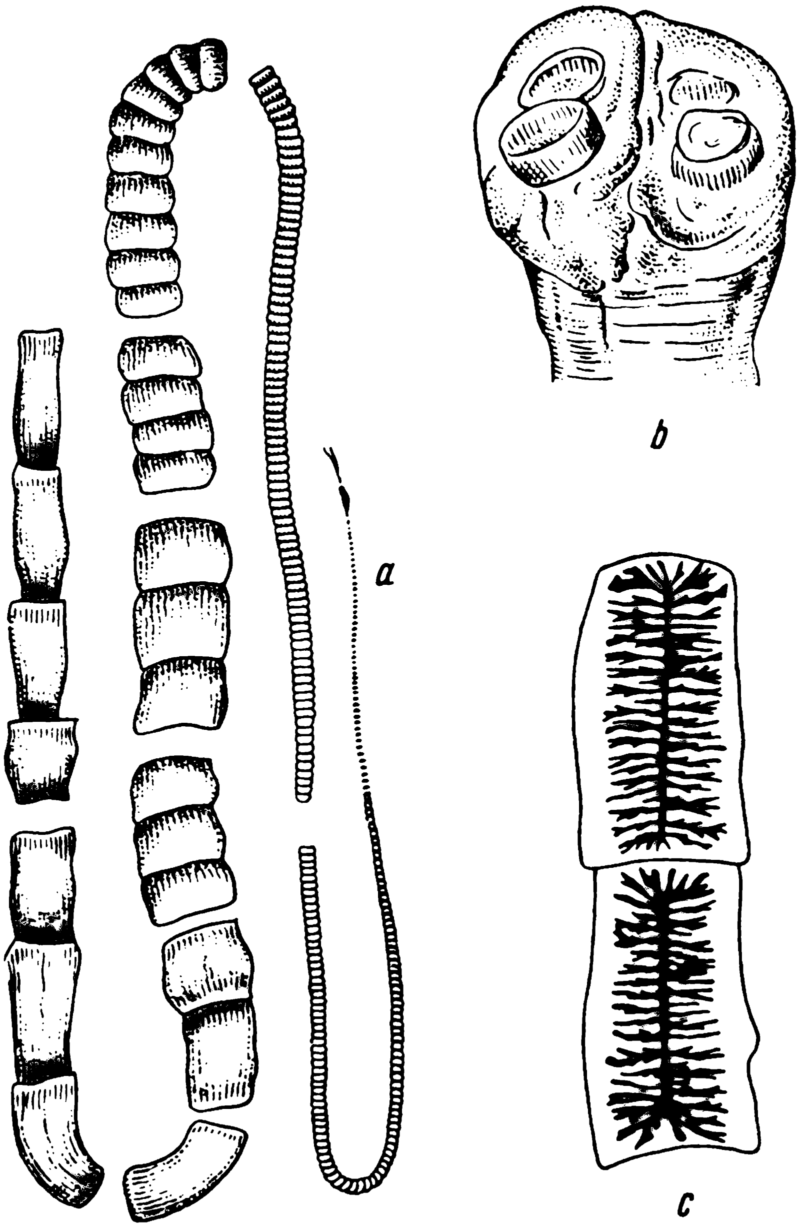


Fig. 65. Beef tapeworm
a—life-size picture of scolex; b—enlarged picture of scolex; c—enlarged picture of proglottides

faeces they disintegrate in the external environment, a large number of oncospheres being liberated from the uterus. Oncospheres contaminate grass with which they are ingested by cattle and, migrating from the intestine into the blood stream, are distributed through the organs and systems of the host. Lodged within the muscular tissue, the embryo develops to the larval stage known the cysticercus which is a bladder-like cyst the size of a pea and contains the worm head (scolex) with suckers. When infected beef is digested the scolex is freed and attaches itself to the intestinal wall.

Clinical picture. Beef tapeworm disease is characterized by a neurodyspeptic syndrome: irritability, increased excitability, and hunger pains in the epigastrium. The disease often runs its course with unmarked symptoms. The principal complaints are those of increased appetite and weight loss. Hypochromic anaemia and eosinophilia are encountered sometimes. The diagnosis is made by detecting segments

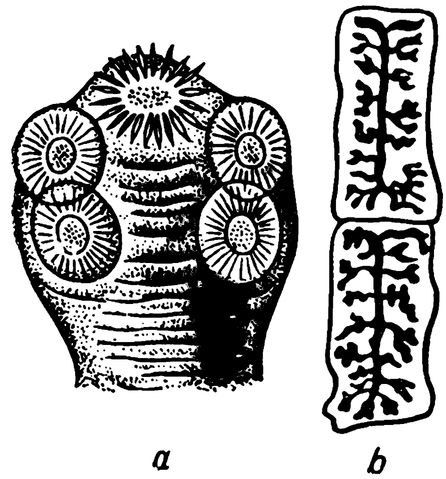


Fig. 66. Pork tapeworm
a—scolex; *b*—proglottides

and eggs in the faeces as well as on the basis of the patient's complaints of a spontaneous discharge of segments.

Taenia solium infestation. Taenia solium, the pork tapeworm, usually manifests itself as a parasite of man by inhabiting the intestinal lumen (Fig. 66). This disease more frequently occurs in regions in which it is the custom to eat raw and slightly salted pork and bacon. The adult pork tapeworm measures about 2-3 m in length. Man becomes infected following ingestion of raw, undercooked or undersmoked pork or bacon, or contaminated meat of wild boar. The life cycle of the pork tapeworm is identical with that of the beef tapeworm, the former's larval stage, however, may develop both in the hog and man, producing a severe condition known as cysticercosis. This usually occurs when detached segments of the parasite are returned to the stomach by reverse intestinal peristalsis. Here the segments are destroyed by digestion and the released oncospheres bore through the wall of the small intestine and are carried by blood and lymph to all parts of the body. Cysticerci are mostly localized to the eye, brain, spinal cord, the skin and subcutaneous cellular tissue.

Clinical picture. The clinical manifestations of *Taenia solium* infestation resemble those associated with *Taenia saginata* infection. The patient complains of debility, headaches, dizziness and reduced working capacity. He is readily excitable and is discomforted by nausea, vomiting, loss of appetite, diarrhoea or constipation. The course of cysticercosis varies with the localization of the parasites. When the latter are lodged in the eye there is a disorder of vision, while cysticercosis of the brain is attended by intense paroxysmal headaches with vomiting and sometimes epileptic fits. Hallucinations and delirious states are also observed, though rarely. The diagnosis of pork tapeworm disease is made by finding oncospheres or proglottides of the tapeworm in the faeces. The patient's evidence of spontaneous passage of tapeworm segments is also important. An essential diagnostic procedure in cysticercosis is the test for fixation of the complement in the patient's blood and cerebrospinal fluid with cysticercosis antigen. Cysticerci in the subcutaneous cellular tissue and muscles are detected by biopsy or x-rays, and those in the eye, by ophthalmoscopy.

Hymenolepiasis (hymenolepidosis) is mainly encountered in children of preschool age, primarily those living in warm regions. It is caused by the dwarf tapeworm *Hymenolepis nana* inhabiting the small intestine. An invasion by this parasite is, as a rule, characterized by the presence of many adult worms in the host's intestine. The worm measures about 3 cm in length. Within two weeks of infecting the lower third of the intestine the oncospheres develop into adult worms which damage the intestinal wall. The entire developmental cycle (from the larval stage to the adult worm) occurs in the body of one and the same host. Dwarf tapeworm disease is contracted from infected humans who spread the infection to other persons and reinfect themselves. The disease is acquired when eggs of this parasite find entrance into the body.

Clinical picture. The patient complains of nausea, vomiting with a copious discharge of mucus, sometimes blood-streaked, abdominal pains, diarrhoea and neurological and mental disorders. Children sometimes suffer from epileptic fits.

Treatment and prophylaxis of cestodiasis. Diseases caused by fish tapeworm, beef tapeworm, pork tapeworm, and dwarf tapeworm are most efficaciously treated with the ether extract of aspidium (Extr. Filicis maris). It is contraindicated for children and emaciated old persons, for active forms of tuberculosis, circulatory failure, gastric and duodenal ulcer, diseases of the liver, kidneys, and in pregnancy. For this treatment to be effective the following prerequisites are necessary: (1) the patient is put on a semiliquid diet poor in fat and rich in carbohydrates 1-2 days before the beginning of treatment; (2) alcohol is forbidden; (3) a saline purgative is administered on the previous day and a cleansing enema is given an hour before taking the preparation. The diet and purgatives are required for a maximal exposure of the head of the parasite; (4) the administration of aspidium is in 1-1$^1/_2$ hours followed by taking a saline purgative (but not castor oil). An enema is applied if no stool has occurred within two hours of administering aspidium; a lunch is allowed not earlier than 1-1$^1/_2$ hours after the purgative has taken effect. Aspidium (in gelatin capsules) is administered in the morning on an empty stomach during 30 minutes in a total dose of 4-6 g divided into 0.5 g doses to be taken every three minutes and washed down with a 1 per cent sodium bicarbonate solution. Many authors believe that a better effect is produced by a single injection of 3-4 g of the preparation directly into the duodenum through a duodenal tube, in the form of an emulsion or a 50 per cent ether solution. Purgatives are introduced through the same tube half-an-hour after the administration of aspidium (save for pork tapeworm disease to avoid vomiting and autoinfection). Poisoning with aspidium produces severe cyanosis, cardiac failure, nausea, vomiting, bloody diarrhoea, headaches and dizziness. The patient should have his stomach lavaged immediately and given a saline purgative, a cleansing enema, and drugs stimulating the heart's action

In the presence of contraindications for the use of aspidium, treatment with pumpkin seed is prescribed. Two days before this the patient is transferred to a light diet without any coarse cellulose. During this two-day period he is given a cleansing enema in the morning and a purgative in the evening. A cleansing enema is also administered on an empty stomach in the morning of the day of treatment; 300 g of hulled seed is ground in small portions in a mortar which after the final portion is washed out with 50-60 ml of water. The ground seeds are then mixed with the water from the mortar and 50-100 g of honey or jam are added. This mass is taken by the patient in small portions during an hour. Four hours later a saline purgative is administered, and after another half-hour, an enema, irrespective of the intestinal response. A breakfast is allowed after the purgative has taken effect.

Tapeworm diseases are treated also with carbon tetrachloride and acrichine (quinacrine).

For dwarf tapeworm infection it is better to administer 3 g of aspidium at 10-day intervals, i.e., during a period covering the entire life cycle of this parasite. This is necessary in view of individual parasites reaching maturity at different times and the likelihood of autoinfection. In addition, before administration of the drug the patient is given methylene blue for three days in daily doses of 0.3 g to be taken in three portions before a meal and, after each course of treatment, yatren in a dose of 1.5 g to be taken three times daily during a period of four days. Hymenolepiasis is also treated with acrichine administered on an empty stomach in doses of 0.1-0.2 g every 5-10 minutes, the daily dose, however, being not larger than 0.8-0.9 g. Half-an-hour after the total dose has been administered a saline purgative is given. The patient is prepared for acrichine therapy in the same way as for treatment with aspidium. Acrichine is contraindicated for renal diseases and mental derangements.

Prophylaxis of taeniasis consists in detection and anthelminthic treatment of all persons invaded by the parasites, enforcement of measures against a possible dissemination of eggs in the external environment and against parasites gaining access to animals on pastures and farmyards, and prevention of human infection by emphasis on personal hygiene; care should be taken to eat only thoroughly cooked beef, pork, and fish.

Trematodiasis. *Opisthorchiasis* is a disease caused by the bloodsucking flat worm known as the cat fluke. It is prevalent in regions where it is the custom to eat quick-frozen raw fish or undersalted fish. The cat fluke, otherwise called Siberian fluke (*Opisthorchis felineus*), is parasitic in the liver of the dog, cat, fox, polar fox, sable, and man. Its first intermediary host is the mollusc, the second is fish, such as roach, ide and tench, carrying encysted young flukes. The mollusc ingests eggs which hatch within its body and release cercariae. The latter leave the intermediate host and freely move about in water. They bore into the skin and muscles of fish and develop into metacercariae. When fish infected with metacercariae is ingested by the final host, their outer shell is digested in the latter's stomach and the inner thin coat, in the duodenum. The young flukes are released, invade the gallbladder and liver and after a month deposit eggs.

Pathologic anatomy. The liver is diminished in size and knobby, the bile ducts are filled with thick dark bile. The gallbladder is distended and filled with a yellow-greenish fluid and black-coloured concrements of loose consistency containing fluke eggs. In certain cases necrosis and cirrhosis of the liver and pancreas and an inflammation of the bile duct walls occur.

Clinical picture. Opisthorchiasis is a chronic disease sometimes running a completely asymptomatic course. The first clinical manifestations may appear a long time after infection. The disease runs its course with symptoms of hepatitis, cholecystitis, cholangitis, pancreatitis, gastrointestinal dyspepsia and dyskinesia of the bile ducts. The patient usually complains of dull pains in the right hypochondrium, sometimes of attacks of hepatic colic, abdominal pains with diarrhoea, occasionally of pains associated with meals and resembling those occurring in gastric and duodenal ulcer. The disease is attended with periodic relapses due to an emotional upset, physical overstrain and chilling. With progression of the disease relapses become more frequent and are less amenable to therapy. An aggravation of the course is often accounted for by involvement of other organs and systems, development of irreversible pathologic changes in the affected organs and sometimes by a reinvasion.

The common symptoms are dizziness, headaches, debility and loss of weight. Sometimes a characteristic yellow colour of the skin and a slight puffiness of the face are encountered. In certain cases nausea, salivation, eructation and heartburn are also complained of. In the majority of cases the appetite is retained, the patient only feeling an aversion to raw fish and meat. Many sufferers have constipation or diarrhoea or both occurring alternately. Dullness of cardiac sounds and hypotension are frequent. The liver and spleen are enlarged.

Bile obtained through a duodenal tube is green in colour and contains epithelial cells, crystalline and amorphous formations. A reduced or absent secretion is often revealed on examination of the gastric juice. Hypochromic anaemia, leucopenia and eosinophilia are constant findings.

Treatment. Antimony and hexachloroethane preparations are the commonly used remedies. Sodium antimony tartrate is prescribed in the form of a freshly prepared sterile 1 per cent solution in a 5 per cent glucose solution with a physiologic salt solution (Stibio-natrii tartarici 1.0, Glucosae 5.0, Sol. Natrii chlorati physiologii 0.85%—100 ml).

The solution is administered intravenously, at a very slow rate, in doses of 10 ml two times daily for three days running. A total of 60 ml of the solution, i.e., 0.6 g of the preparation is administered in one course of treatment. Hexachloroethane is also very effective against opisthorchiasis. The drug is prescribed in gelatin capsules to be taken per os, in doses of 12-40 g during one course of treatment for adults. The 24 hour dose is 6-10 g. One course of treatment lasts 2 days. The interval between courses is from four to six weeks. Control over the efficacy of therapy is carried out by examining the duodenal juice and counting the number of fluke eggs in the faeces before and after treatment during a period of three months and longer. Specific therapy must be combined with other measures, such as duodenal drainage with lavage of the bile ducts, use of cholagogues, polyvitamins, bromides. When inflammation of the biliary passages is suspected, diathermia, ozocerite and UHF are applied to the hepatic region and antibiotics are prescribed.

Diseases of the Liver and Bile Ducts

AN OUTLINE OF ANATOMY AND PHYSIOLOGY

The liver weighs 1,500 to 1,800 g. It is situated in the right hypochondrium, and is held in place by several ligaments but mainly by force of the intra-abdominal pressure. In the superior and posterior aspects the liver adjoins the diaphragm and is partly contiguous to the anterior abdominal wall. The posterior-lower concave surface lies upon the intestine, and adjoins the right kidney and adrenal gland. The liver consists of four lobes. Between the right and left lobes of the liver there is a recess lodging the gallbladder. The latter is pear-shaped, measures 8-10 cm in length and holds 30-40 ml of bile. Its body lies upon the transverse colon and duodenum.

The hepatic cells adjoin one another, forming the hepatic lobules; the latter are grouped to form lobes interconnected by mesenchymal tissue lodging the Kupffer cells of the reticuloendothelial system. These cells have extensions to every hepatic cell. Between the hepatic cells there are the bile canaliculi which merge into the interlobular bile ducts. The latter give rise to two bile ducts which blend into the hepatic duct (ductus hepaticus) giving off a branch known as the cystic duct (ductus vesicae fellae) to the gallbladder. The further portion of the hepatic duct is known as the common bile duct (ductus choledochus) which enters the duodenum in the middle of its descending part near the ampulla of Vater and the pancreatic duct (Fig. 67).

The transverse fissure (porta hepatis) of the liver receives the portal vein and the hepatic artery. The portal vein brings to the liver venous blood from the organs of the abdominal cavity and branches off into the interlobular venules terminating in capillaries. The latter communicate with the capillary network of the hepatic veins passing within a lobule. The capillaries of the hepatic veins widely ramify and enter the inferior vena cava. The hepatic artery supplying the liver ramifies into capillaries going between the lobules and also joining the central vein and through the latter, the inferior vena cava. Kupffer's stellate cells are arranged along the course of the vessels. They play a highly important role in the secretion of bilirubin and are capable of phagocytosis of foreign substances. The liver is innervated by the sympathetic and parasympathetic nervous systems. The nerves branch off from the solar plexus, enter the liver together with the hepatic artery and ramify along the course of the vessels. Microscopy of hepatic cells reveals pigment granules and glycogen lumps, globules of fat also being a frequent finding.

The liver performs highly important and intricate functions essential to human life, and is, as Pavlov aptly put it, the principal laboratory of the human body. The liver performs external and internal secretion and is concerned with metabolism.

External secretion. This function is essentially one of secreting bile, which is produced continually and is delivered into the duodenum and the gallbladder. The bile constituents include bile pigments, bile acids and cholesterol. Bile secretion is elevated after ingestion of water, salicylates, atophan, egg yolk, meat, bread, and milk. Other cholagogic agents are pituitrin, histamine, magnesium sulphate, calomel, etc.

The gallbladder wall lodges smooth muscle fibres with a well-developed system of nerve cells. The vagus nerve is the motor nerve of the gallbladder and causes the latter's contraction, while the sympathetic nerve produces a relaxation of its wall. Expulsion of bile from the gallbladder in which it accumulates and becomes inspissated occurs when food has entered the stomach and duodenum, as a result of contraction of the gallbladder and opening of Oddi's sphincter situated at the end of the common bile duct. Secretion of bile into the duodenum may also be provoked by introducing magnesium sulphate into the latter or by a subcutaneous injection of pituitrin. The gallbladder is capable of absorbing water from its bile contents owing to which the cystic bile is dark and thick in consistency.

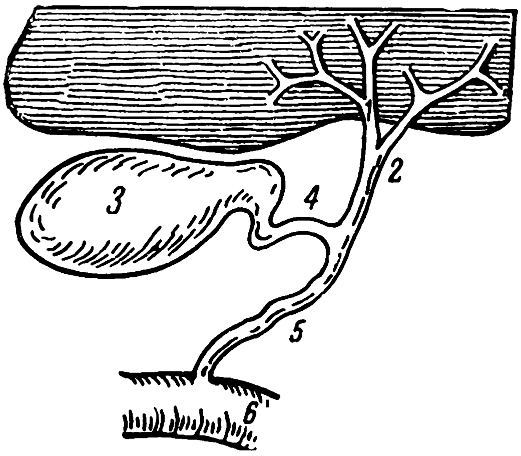


Fig. 67. Biliary passages

1—intrahepatic bile ducts; *2*—common hepatic bile duct; *3*—gallbladder; *4*—cystic duct; *5*—common bile duct; *6*—duodenum

For a diagnostic study of gallbladder function and examination of cystic duct patency duodenal drainage is employed. In this procedure, 50 ml of a 25 per cent magnesium sulphate solution is introduced into the duodenum through a tube. Duodenal drainage may help obtain bile passed from various parts of the bile tract. In view of this, portions A, B, and C are distinguished in bile obtained by drainage. A bile is a specimen of duodenal contents with an admixture of bile; this portion is obtained when the olive attached to the end of the tube enters the duodenum. B bile obtained within 5-10 minutes of injecting magnesium sulphate is cystic bile and is of a dark colour and thick consistency. C bile is from the hepatic ducts and has a pale amber colour.

During an inflammatory process the cystic bile may show the presence of leucocytes, mucus, epithelial cells, bacteria, parasites, cholesterol crystals, etc. In occlusion of the cystic duct cystic bile fails to appear, and in occlusion of the common bile duct no bile is conveyed into the duodenum, therefore, A bile cannot be obtained.

Bilirubin is produced by the blood pigment haemoglobin at the sites of destruction of erythrocytes in the reticuloendothelial tissue (spleen, liver, etc.). Bilirubin circulating in the blood is bound up with protein; in the liver, however, it loses its protein and is secreted in pure form. The normal bilirubin content in the blood is 0.3-0.5 mg/100 ml. In pathology the amount of bilirubin is considerably increased to reach the level of 10-15 mg/100 ml. Bilirubin bound up with protein (indirect) as well as protein-free bilirubin (direct) is determined by means of a diazo reagent. This test essentially consists in the appearance of a pink colour on addition of the reagent to blood serum. In cases when a pink colour appears immediately upon addition of the reagent, the reaction is known as immediate direct, which is evidence of mechanical jaundice. If, however, a pink coloration appears with a delay of 2-3 minutes a delayed direct reaction is said to be taking place; it occurs in parenchymatous jaundice. A violet-pink colour appearing after alcohol has been added in order to settle protein is evidence of an indirect reaction which occurs in haemolytic jaundice.

In addition to bilirubin and its isomer biliverdin the secreted bile also contains bile acids which are produced in the liver itself, heterogenous substances, various bacteria, and dyes. The latter property is used in cholecystography.

About 1 litre of bile is supplied into the intestine in 24 hours. In the intestine, bilirubin is reduced to urobilin or stercobilin. Part of the latter is spent on colouring the faeces and is eliminated from the intestine in the stool. The bulk of urobilin is again conveyed to the liver in which it is oxidated and turned into bilirubin. A diseased hepatic cell is unable to convert into bilirubin all the urobilin absorbed from the intestine. In such a case part of the urobilin is carried away with the blood in which it accumulates and is excreted with the urine. Consequently, the presence of urobilin in the urine points to a failure in the function of the liver. An exception is noted with an excessive urobilin production in the body, as is the case in haemolytic jaundice.

The metabolic function of the liver is extremely diversified. Products of protein, fat, and carbohydrate digestion are absorbed from the gastrointestinal tract by the liver in which they undergo further chemical processes. Complex carbohydrates (starch, polysaccharides) exposed to the action of the salivary enzyme ptyalin and the pancreatic enzyme diastase are split into monosaccharides. The latter are absorbed into the blood and brought to the liver, where they are converted into glycogen under the influence of insulin and are deposited in the hepatic cells. Normally glycogen accounts for 5-10 per cent of the total weight of the liver. Whenever necessary, glycogen is converted into glucose which is supplied to the blood to meet the energy requirements. The blood glucose content on an empty stomach ranges from 80 to 120 mg/100 ml. It increases after ingestion of food, especially that rich in carbohydrates, and after physical or mental strain. Glycogen deficiency in the liver entails the latter's fatty degeneration and an impairment of the body resistance to infections, poisons, and intoxications. And conversely, an increase in the amount of glycogen in the liver serves to fortify the body resistance. Several tests are used to examine the glycogen-forming function of the liver: the glucose tolerance test, the galactose tolerance test, etc. The glucose tolerance test is carried out as follows: after determination of the blood sugar level on an empty stomach the patient is given 100 g of glucose in a glass of water, following which the sugar content of the blood is determined every half-hour during two hours. Normally the amount of sugar in the blood after the test shows a small increase, and reaches the initial level by the end of the 2nd hour. With a defective carbohydrate metabolism in the liver, the sugar content of the blood is considerably elevated after the test, being twice as high as the initial level, and fails to return to this level by the end of the 2nd hour.

The galactose tolerance test is simpler and requires no special skill. The patient is given 40 g of galactose in a cup of tea in the morning on an empty stomach, after which urine is collected during six hours, and its galactose content is determined. Normally the urine must contain not more than 2 g of galactose after a test; a higher content indicates a disturbance in the carbohydrate metabolism in the liver.

The role of the liver in protein metabolism. Protein substances exposed to the action of pepsin and, in the intestine, to that of trypsin and erepsin are split into amino acids and are carried to the liver through the portal vein. A considerable part of amino acids is utilized in the formation of proteins, another part is converted into glucose. Some amino acids are converted into urea. In hepatic dysfunction the formation of protein is disturbed, the level of amino acids in the blood is considerably elevated, with a corresponding decrease in the amount of urea in the urine. The liver exhibits its protein-forming function by taking part in the production of albumins, fibrinogen and prothrombin. A disturbance in the normal production of albumin gives rise to oedema, and that in fibrinogen and prothrombin output, to predisposition to bleeding.

The role of the liver in fat metabolism. Digestion of fats and their absorption in the intestine take place in an indispensable presence of bile acids. Therefore, cessation of bile supply to the duodenum results in malabsorption of fats. In hepatic insufficiency, the splitting of fatty acids fails to reach its final stages—water and carbon dioxide—and is delayed in the stage of acetone bodies (acetone, aceto-acetic acid, and β-oxybutyric acid). The liver is the principal site of cholesterol production. Normally the serum cholesterol content is 120-180 mg/100 ml. An affection of the liver is accompanied by a reduction in this content. This is partly due to malabsorption of fats from the intestine since the latter supplies material for cholesterol formation.

The antitoxic properties of the liver. The liver destroys toxic substances usually formed in the intestinal tract as well as heterogenous poisons introduced into the body from without. The detoxification function of the liver safeguards man against many diseases. Pavlov attached tremendous importance to this antitoxic function. He wrote that for the human body the liver is the most vigilant guard which keeps in check the toxic substances arriving from the digestive tract and renders them harmless to other organs.

The liver is very rich in vitamins as well as in iron.

SYMPTOMS OF DISEASES OF THE LIVER AND BILE DUCTS

When taking the patient's medical history emphasis should be laid on an account of his working and living conditions, dietary habits, a history of past diseases, particularly of the gastrointestinal tract, and the condition of the nervous and endocrine systems, because a hepatic disease is often directly associated with the above-mentioned factors. For example, overeating, particularly of fatty foods, alcoholism, and a sedentary way of life may sometimes suggest a diagnosis of fatty degeneration of the liver. A persistent affection of the liver is encountered after Botkin's disease and in chronic infections, such as syphilis, tuberculosis, and malaria. Involvement of the liver and bile ducts is often found after enteric fever, gastritis, and gastroenteritis. The patient's complaints of pruritis, abdominal distention, meteorism, loss of weight, and pains in the right hypochondrium facilitate identification of diseases of the liver and bile ducts. Among the characteristic signs of a hepatic disease are an icteric colour of the skin, sclera and of the mucous membranes accessible to view, bulgings in the right and left hypochondrium, ascites, dilatation of the subcutaneous veins around the navel known as caput medusae, scratches on the skin, etc.

Palpation and percussion of the liver and spleen may yield valuable evidence for a diagnosis. The size of the liver (enlarged or contracted), consistency (firm, soft), features of its surface (uneven, nodular), the condition of the lower border (blunt, sharp) are important diagnostic findings.

Jaundice (icterus). This disease is due to the presence of an excessive amount of bilirubin in the blood and tissues. It may be obstructive (mechanical), hepatogenic (parenchymatous), and haemolytic.

Obstructive jaundice is caused by obstruction of the common bile duct with a calculus or helminths, compression of the bile ducts with a malignant newgrowth, gumma, enlarged lymph nodes of the transverse fissure, etc. The secretion of bile continues but the latter fails to pass into the duodenum and is collected before an obstacle, rupturing the bile canaliculi and entering lymphatic and blood vessels. In obstructive jaundice the bilirubin content of the blood is elevated as early as in 24 hours, and after another 2-3 days an icteric coloration of the soft palate, mucous membranes, and the skin appears. Later, the skin acquires an olive colour and sometimes a greenish hue. The urine darkens and takes the colour of dark beer with a bright-yellow foam. Because of an arrest of the flow of bile to the duodenum the faeces become discoloured, acquire a clayish hue and contain no stercobilin. The serum bilirubin exhibits a direct reaction to a diazo reagent. In obstructive jaundice bile acids and cholesterol are accumulated in the blood in addition to bilirubin. Retention of bile acids causes pruritis and bradycardia. Haemorrhagic diathesis, predisposition to bleeding, is due to a deficit of prothrombin and partly fibrinogen in the blood. In prolonged obstructive jaundice congestion of bile causes damage to the hepatic cells and a disturbance in the function of the liver.

In *hepatogenic jaundice* bile fails to pass either into the duodenum or the gallbladder. This is due to infection or intoxication which damage the liver to the degree of destruction of its cells along with the bile canaliculi, as a result of which bile enters the blood stream and impregnates the tissues. Hepatogenic jaundice occurs in Botkin's disease, parenchymatous hepatitis, cirrhosis and dystrophy of the liver, etc. In hepatogenic jaundice the blood bilirubin content is elevated, the blood exhibiting a delayed direct reaction to a diazo reagent. The amount of cholesterol in the blood is reduced because of decomposition of hepatic cells in which cholesterol is produced, and partly due to inadequate conveyance of fats to the liver. The faeces are discoloured only when the disease is at its height. Later, with restoration of hepatic function and bile supply to the duodenum, the faeces begin to take on colour and are found to contain stercobilin. Only bilirubin is present in the urine at the peak of the disease. When jaundice begins to subside urobilin also appears in the urine and for a certain period gradually increases in content in proportion to the entry of bile into the intestine in the presence of a still insufficient function of Kupffer's cells of the reticuloendothelial system.

Later, with a progressive improvement, the urobilin content of the urine declines to the degree of its total disappearance corresponding to the complete restoration of the function of the hepatic and Kupffer's cells. The presence of urobilin indicates, on the one hand, that bile has begun to pass into the intestine, and on the other hand, that hepatic function is insufficient since the liver is unable to take up the urobilin and convert it into bilirubin, as a result of which the former enters the blood stream and is passed with the urine. Hepatogenic jaundice is accompanied by a disturbance of all functions of the liver—carbohydrate, protein, and fat metabolism and antitoxic activity. A disturbance in the carbohydrate metabolism is confirmed by the galactose tolerance test, that in the protein metabolism, by a considerable increase in the amino acid levels of the blood and urine and a diminution of the amount of urea in the urine. Severe forms of hepatogenic jaundice are accompanied by cholaemia and sometimes terminate in coma.

In *haemolytic jaundice* the patient is rather pale than icteric in the face. No itching of the skin is complained of. All functions of the liver are preserved; it is not enlarged and exhibits no anatomic or histologic changes. Jaundice results from a diminished resistance of erythrocytes, their intensified decomposition, and formation of a considerable amount of bilirubin. In haemolytic jaundice serum bilirubin shows an indirect reaction to a diazo reagent, because it is bound up with protein (extrahepatic bilirubin). In spite of its considerable production, the amount of bilirubin in the blood is fairly low, because almost all of it is conveyed to the liver and intestine in a total quantity considerably larger than normal. Its excess in the intestine lends the faeces a dark colour, while the urine shows an increase in its urobilin content, since the liver is unable to retain all the urobilin and convert it into bilirubin. No bilirubin occurs in the urine. It is supposed that this absence is due to its being bound up with large protein molecules and not capable of passing through the glomerular filter.

Cholaemia and hepatargia (severe hepatic insufficiency). Severe diseases of the liver produce autointoxication of the body manifested by haemorrhages, nervous and mental disorders, and hepatic coma. This intoxication is essentially due to decomposition of hepatic tissue. In cholaemia the amount of amino acids is considerably increased while that of uric acid is correspondingly reduced. Autointoxication of the body in cholaemia is mainly caused by products of intestinal putrefaction which in hepatic insufficiency fail to be completely detoxified.

Cholaemia occurs with acute and subacute dystrophy of the liver and in cases of obstructive jaundice complicated by liver necrosis.

Portal hypertension. All processes in the liver which are accompanied by a disturbance in the intrahepatic blood flow cause an elevation of pressure in the portal venous system. Portal hypertension is also encountered in diseases of the portal vein itself, in inflammations of its trunk, and in thrombosis of the hepatic veins at the site of their entry into the inferior vena cava.

Portal hypertension often results from a failure of the right cardiac ventricle in adhesive pericarditis. It develops slowly or acutely. Acute portal hypertension occurs in thrombosis of the trunk of the portal vein or after a severe contusion of the abdomen. The symptoms of portal hypertension are as follows: (a) enlargement of the spleen; (b) ascites; (c) development of collateral circulation.

While the above-mentioned clinical symptoms appear gradually and are of a chronic character in cirrhosis of the liver, in acute forms of portal hypertension they develop within a few hours and early cause the patient's death.

EXAMINATION OF THE LIVER AND SPLEEN

The liver is a compact airless organ. A lung is situated above the liver, the stomach, to the left, and the intestine, below.

Percussion of the liver elicits a dull sound only in that part which directly adjoins the thoracic and abdominal walls. In the other parts the hepatic sound combines with that elicited from the adjacent hollow organs. The liver has two upper boundaries of dullness: the relative and the absolute. The latter corresponds to the part of the liver not covered with the lungs. The relative boundary of liver dullness begins at the level of the diaphragm. Its upper boundary coincides with the lower boundary of the lungs and lies at the level of the 6th rib along the midaxillary line.

It is difficult to determine the lower boundary of the liver by percussion because of the adjoining stomach and intestine. The lower boundary of the liver extends from the midaxillary line, along the 10th rib, crosses the midclavicular line, passes along the costal arch, and to the median line on the border of the upper and middle third of the distance between the navel and the base of the xiphoid process. At left the area of hepatic dullness is coalescent with the area of absolute cardiac dullness.

The liver boundaries must be determined by a slight percussion, having in mind the surrounding air-filled organs. The size of the liver is determined after finding the upper and lower boundaries. Along the midline the liver measures 6-8 cm; along the midclavicular line, 9-11 cm, and along the midaxillary line, 11-12 cm. The hepatic boundaries, both the upper and the lower, may be depressed in pulmonary emphysema, pneumothorax and pleurisy because of the diaphragm being in a low position. In pregnancy, meteorism, ascites and tumours in the abdominal cavity the upper boundary is elevated. Absolute hepatic dullness is increased in hypertrophic cirrhosis of the liver, abscesses, hydatid cyst, cancer, and in inflammation of the liver. In atrophic cirrhosis and in acute dystrophy

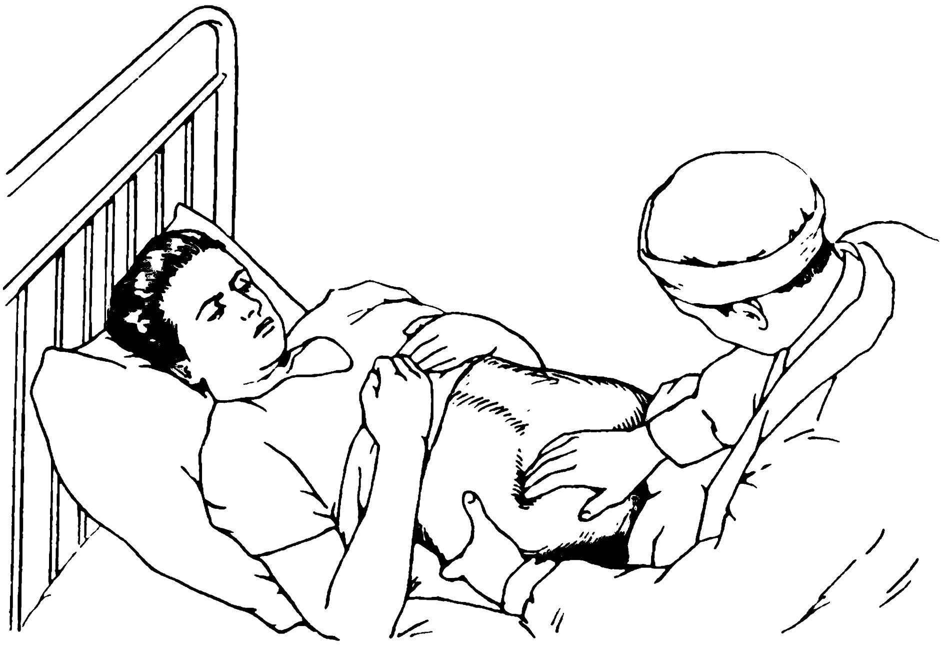


Fig. 68. Palpation of the liver

of the liver the boundaries of absolute hepatic dullness are sharply contracted because of a diminution of the liver itself. In perforation of the stomach there is a clear tympanitic resonance in the area of absolute hepatic dullness as a result of escape of air into the space between the diaphragm and the liver.

Palpation of the liver. For palpation of the liver the patient is put in a recumbent position with relaxed abdominal muscles. The examiner determines the lower border of the liver (sharp, blunt), its size and consistency (firm, soft), and the character of its surface (smooth, nodular). The examiner's hands should be dry and warm, and the nails cut short. Palpation is performed without pressure after determination of the lower hepatic boundary by percussion. The right hand is placed flatwise on the right upper quadrant of the abdomen, while the region of the liver is supported from behind with the palm and four fingers of the left hand. The thumb of the left hand is pressed to the rib margin in order to reduce the volume of the abdominal cavity and limit the excursion of the chest wall (Fig. 68). Taking advantage of every exhalation during which the abdominal muscles are relaxed, the liver is palpated with the right hand. During an inhalation the lower border of the liver glides into a space formed when the right hand is pressed to the abdominal wall and, at the same time, moved further upward, then the liver escapes out of this space, goes down, bypasses the finger tips and slips behind them. Usually light palpation of the anterior surface of the liver is performed in the same way. In healthy persons the lower border of the liver is even, soft, and painless. In venous congestion it is rounded; in cirrhosis, firm; in tumours, nodular; in amyloidosis, smooth and extremely firm. The liver has an uneven surface in syphilis, hydatid cyst, and newgrowths of this organ.

Percussion of the spleen. The spleen is normally situated in the left hypochondrium along the midaxillary line between the 9th and 11th ribs. Its anterior border does not extend beyond the anterior axillary line. Posteriorly, the area of spleen dullness is coalescent with that of the muscles and kidneys. The width of spleen dullness is 4-8 cm. The borders of spleen dullness are determined by slight percussion, the patient lying on his right side, because the spleen lies between air-filled organs which give out a tympanitic resonance upon percussion.

Palpation of the spleen. The patient is told to lie on his right side or on his back. Palpation is performed with half-bent fingers of the right hand applied perpendicularly to the rib margin in the region of the 10th rib when the patient inhales deeply and the edge of the spleen approaches the fingers. The examiner determines the consistency and tenderness of the spleen and may palpate the splenic notch in considerable spleen enlargement. The spleen is enlarged and protrudes from under the left hypochondrium in many diseases, including infections (syphilis, tuberculosis, malaria), cirrhosis of the liver and leukaemia. In malaria and leukaemia the spleen sometimes fills almost the whole of the abdominal cavity.

In inflammation of the serous coat of the spleen palpation of this organ is painful.

Hepatomegalia and hepatosplenomegaly. In a number of diseases of the liver and adjacent organs—cancer, hydatid cyst, and cirrhosis of the liver, adhesive pericarditis and diseases of the heart, particularly of the right ventricle, the liver is enlarged to considerable size; hence the term hepatomegalia. Most commonly a concomitant parallel enlargement of the liver and spleen is observed, i.e., the so-called hepatosplenomegaly. Their concurrent involvement is accounted for by the fact that they are both rich in reticuloendothelial tissue, and in addition, they have a circulatory system in common: from the spleen the blood enters the system of the portal vein, which results in toxic substances being delivered from the spleen to the liver. Hepatosplenomegaly is encountered in syphilis, malaria, brucellosis, subacute bacterial endocarditis, haemolytic jaundice, pernicious anaemia, conditions following transfusions of incompatible blood, leukaemia, erythraemia, amyloidosis, etc.

HEPATITIS

Hepatitis is a term denoting an acute or chronic inflammation of the liver. In this disease an inflammation of the hepatic cells is concurrent with reactive changes in the interstitial tissue. In respect of its course hepatitis may be acute or chronic, and for the site of involvement, diffuse or focal. Diffuse parenchymatous hepatitis is attended with jaundice, whereas focal hepatitis is anicteric. Jaundice is also absent in those forms of hepatitis in which inflammation is mainly confined to the interstitial tissue. Interstitial hepatitis usually occurs in malaria, syphilis, brucellosis, subacute bacterial endocarditis, etc.

Acute hepatitis occurs in the following forms: (1) primary infectious, or diffuse, hepatitis with a primary involvement of the liver in one or another infection, as is the case in Botkin's disease, Weil-Vasilyev's disease, and tropical yellow fever; (2) secondary infectious hepatitis with a secondary involvement of the liver in an infectious disease, as is the case, for example, in lobar pneumonia, relapsing fever, paratyphoid; (3) toxic hepatitis due to the effects of various poisons.

BOTKIN'S DISEASE

Botkin's disease, otherwise known as epidemic or infectious hepatitis, is an acute viral disease of a wide distribution. At Virchow's suggestion this disease was termed catarrhal jaundice since he believed it to be due to obstruction of the common bile duct with mucus during inflammatory processes in the duodenum. S. Botkin, however, in 1880 advanced the idea of an infectious origin of this disease and adduced weighty arguments to prove it (involvement of not only the liver but also of the nervous system, the kidneys, enlargement of the spleen, etc.). Nevertheless, it was not before 1940 that the term "Botkin's disease" was introduced into medical usage owing to the efforts made by M. Konchalovsky, an outstanding Soviet physician, to honour the memory of the scientist who was first to uncover the infectious character of jaundice. Botkin's disease occurs sporadically or in epidemic form. It more commonly afflicts children and adolescents, although this disease is fairly frequent in adults as well as in elderly persons. The epidemic form is especially prevalent in wartime, under conditions of crowding and mass-scale migration.

Aetiology and pathogenesis. Botkin's disease is due to a filterable virus present in the blood, liver, and nasopharyngeal washings, and eliminated with faeces and urine. The virus is infective only for man. It is not visible under a conventional microscope and may be discovered only by electron microscopy at 100,000-200,000 magnification. The filterable virus is highly resistant and survives in water, foodstuffs, on objects and hands for days and weeks and, when dried or at room temperature, for several months. When in a frozen state, the virus remains viable for years.

Epidemiology. The source of virus is a sick person who may spread the infection by close personal contact from the final days of the incubation period through the entire course of the disease. The contagium is especially virulent, however, in the prodromal period and in the first week of the disease. The patient's blood is highly contagious even in a dose of 0.1 ml. A highly dangerous source of infection is the asymptomatic virus carrier since the latter feels no indisposition and takes no precautions against infection of other people, members of his family in particular. Infection is acquired by close personal contact as well as through virus-contaminated water and food. Flies are also known to transmit this disease. The virus gains entry to food above all on hands contaminated with a sick person's excreta when visiting a water-closet, handling a telephone or handrails in a bus, trolleybus, tram, etc. Infection usually enters the body by one of the following two routes: through the mouth when eating contaminated food or drinking water, and parenterally during transfusions of blood, plasma and serum, prophylactic vaccinations, and injections performed by means of inadequately sterilized syringes and needles. Infectious hepatitis caused during vaccinations or injections is known as inoculation or serum hepatitis.

Epidemic hepatitis most commonly occurs late in autumn, early in winter, or in spring. The incubation period lasts from 2 to 14 days. The inoculation or serum form of Botkin's disease is encountered all year round and is distinguished by its lower infectiveness and longer incubation period (3 to 11 months), since in view of its lower titre the virus requires a longer time for reproduction. Transmission of infection from mother to foetus is also possible.

Pathogenesis. The virus of Botkin's disease in infection by the oral route enters the gastrointestinal tract, and in infection through inoculation, the blood and lymph. In both cases, however, it gains access to the liver in which it propagates. Botkin's disease causes inflammatory and degenerative changes with decomposition of hepatic cells and damage to the bile calculi as a result of which bile enters the lymph flow and subsequently the blood stream. The reticuloendothelial tissue of the spleen, the gallbladder, and the nervous and endocrine systems become involved simultaneously with the liver. Sometimes allergic manifestations (urticaria, etc.) are observed in connection with absorption of the products of hepatic cell decomposition (proteins) by the blood.

Clinical picture. Between the entry of contagium and the onset of the disease there is a latent period lasting 2-3 weeks on the average. Botkin's disease may run its course with or without jaundice (anicteric form). Anicteric hepatitis runs a mild course and, because of absent jaundice, often remains unidentified and is recognized as influenza or catarrh of the upper air passages. Anicteric hepatitis poses a great danger to health since failure to recognize the disease in due time is likely to contribute to the spread of virus to contacts. The icteric form of Botkin's disease is known to run its course in the following three stages: (a) pre-icteric (prodromal); (b) icteric; (c) convalescent (period of abatement of jaundice).

The pre-icteric stage lasts one to two weeks and sometimes longer. In this period the patient usually complains of indigestion, anorexia, nausea, less frequently vomiting, eructation, heartburn, a feeling of pressure and pains in the right hypochondrium, heaviness and distention in the abdomen, and diarrhoea. These are joined by lassitude, general malaise, debility, irritability, early fatigue on exertion, headaches, sweats, and pains in the heart. There are often pains in the muscles and joints, as well as coughing, rhinitis and insomnia. The temperature rises to 37-38°C. This rise may be brief and subfebrile. A higher fever is accompanied by headaches, conjunctivitis, tracheitis, bronchitis, pains in the muscles and joints, and urticaria. For this reason, Botkin's disease is often mistaken for malaria, bronchitis, rheumatic fever, etc.

In the pre-icteric stage the liver is enlarged and protrudes from under the rib margin by 2-3 cm. It is smooth and tender on palpation. The urine is dark, has a beer-coloured foam and contains bile pigments and urobilin. The spleen is also enlarged sometimes. In the same period, an elevation of the bilirubin content of the blood and sometimes discoloration of the faeces may be encountered. There is an increased activity of aldolase (up to 30-60 U) and transaminase (enzymes produced by decomposition of liver parenchyma). The blood exhibits eosinophilia and monocytosis.

The icteric stage of the disease lasts 2 to 3 weeks. The scleras and later the skin turn yellow. Jaundice tends to increase rapidly, liver enlargement progresses, the faeces are discoloured and contain no stercobilin. The urine exhibits the presence of bile pigments, bile acids, protein and casts, but no urobilin because the bile fails to pass into the duodenum. There is pruritus, a slow pulse, and a drop in the arterial pressure. The stools are fatty and malodourous since in the absence of bile decomposition processes in the intestine are markedly intensified.The bilirubin content of the blood is elevated; there is leucopenia, lymphocytosis, monocytosis, and a reduced ESR, while blood coagulation is impaired since the amount of prothrombin and partly of fibrinogen is diminished. The amount of globulins, especially γ-globulins, is increased, and that of albumins, decreased. The cholesterol content of the blood is also reduced. All functions of the liver—carbohydrate, protein, and fat metabolism, and antitoxic activity—are deranged. A highly characteristic feature of the icteric stage is a nervous and mental disorder. Adynamia, headaches, insomnia, and irritability are common findings. In this period signs may appear of an inflammation of the bile ducts due to a virus or other organisms (*E. coli*, etc.), for which congested bile is a favourable environment.

The convalescent period sets in with a subsidence of liver enlargement and jaundice. Simultaneously there is a general improvement in the patient's condition, an increase in appetite, and abatement of pruritus, while the urine becomes paler and is passed in quantity and the faeces acquire a normal colour. Languor and irritability disappear and sleep becomes normal.

In respect of its course Botkin's disease may be mild, moderately severe, or severe. In mild, or ambulant, forms of Botkin's disease jaundice may be very brief and have almost no effect on the patient's subjective condition. In severe forms of epidemic hepatitis, there is torpor, somnolence, and prostration, while jaundice is highly intense and the liver is enlarged. All functions of the liver are disturbed considerably. Sometimes the patient lapses into a coma the onset of which is signalized by a sudden lassitude, languor, an increase in tendon reflexes, confused consciousness, nausea, vomiting, contraction of the liver, and a continuous drop of the arterial pressure, oedema, ascites and predisposition to bleeding.

In the majority of cases Botkin's disease, including its protracted forms, ends in recovery. In isolated cases, however, it may take a chronic course. The latter is characterized by frequent exacerbations with a severe disturbance in the function of the liver, which fails to recover in a period of remission. The disease may become chronic because of belated hospitalization, a digression from the hygienic or dietary regimen, drinking alcoholic beverages, physical and mental overstrain, as well as intercurrent diseases, such as influenza or gastroenterocolitis. These chronic forms may terminate in death due to hepatic insufficiency.

Botkin's disease may be accompanied by complications: pancreatitis, cholecystitis, cholangitis, acute and subacute dystrophy of the liver, as well as cirrhosis of the liver. The disease runs a more severe course and produces complications in children, pregnant women, and persons with cardiovascular lesions. Usually a person recovered from Botkin's disease is unable to work during 2-3 months.

Treatment and prophylaxis. The patient must be taken to a hospital for infectious diseases and, prior to hospitalization, isolated at home in order that the spread of infection may be avoided. In the initial and acute periods of the disease bed rest is indispensable. The diet should be composed of easily assimilated foods, mainly carbohydrate (gruels, mashed potatoes, sweet compotes, thin jellies, fruit juices, vegetables, etc.). Proteins are given in quantities of 100-120 g per 24 hours as cottage cheese, milk, and boiled meat. The intake of fats (butter, sour cream, milk cream) must not exceed 50 g, because the splitting of fat is impaired. Meals must be frequent and served in divided portions, since food is a potent cholagogic agent. The diet must be enriched with supplements of all vitamins: vitamin K (vicasol in a dose of 0.01 g three times daily), vitamin B_{12} subcutaneously or instramuscularly in a dose of 100-150 γ and vitamin B_6, as well as ascorbic acid (intravenously in a dose of 1 ml of a 5 per cent solution). The intake of liquid may be unrestricted in the absence of ascites and a circulatory disturbance. It is advisable to prescribe alkaline water which stimulates bile secretion. One is recommended an intravenous infusion of a 40 per cent glucose solution which increases the amount of glycogen in the liver, improves the latter's functions, and strengthens body resistance to infection. To facilitate deposition of glycogen in the liver, insulin in a dose of 8-12 U is injected subcutaneously 15-20 minutes before glucose infusion. In moderately severe and in severe cases a 5 per cent glucose solution and a physiologic salt solution are injected not only intravenously but also subcutaneously in doses of 0.5-1.0 litre per 24 hours. To reduce congestion in the bile ducts, duodenal drainage employing a 25 per cent magnesium sulphate solution in a dose of 50 ml is undertaken. This lavage is allowed only in the period of convalescence. For bleeding, blood transfusions in doses of 100-200 ml every other day, vitamin K, as well as intravenous infusions of 10 ml of a 5-10 per cent calcium chloride solution are necessary. The latter solution may also be administered per os in one tablespoonful dose 3-4 times daily. Penicillin in a dose of 1,000,000 U per 24 hours (250,000 U 4 times daily), streptomycin in a dose of 0.5 g twice daily, and levomycetin in a dose of 0.5 g three times daily are prescribed to prevent or eliminate bacterial inflammation in the liver and bile ducts. The cholagogic agents used are salicylates and magnesium sulphate but never atophan in view of its harmful effects on the liver. Fatty infiltration of the liver is prevented with methionine given in a dose of 0.5 g three times daily or 300 g of cottage cheese eaten within 24 hours as well as with lipocaine taken in a dose of 0.3 g three times daily. In isolated cases when the icteric stage takes a prolonged or malignant course, ACTH in a dose of 10-20 U three times daily, prednisolone, or prednisone in a dose of 10 mg 2-3 times daily may be administered in view of their anti-inflammatory and desensitizing action and their favourable effect on metabolism. Pruritus is relieved by taking a cool bath with an addition of soda or vinegar, sponging one's body with vinegar, a 1-2 per cent carbolic acid solution, or a 3 per cent methyl-alcohol solution. For the same purpose 0.5-1.0 ml of a 0.1 per cent atropine sulphate solution as well as dimedrol is injected subcutaneously.

The patient should be discharged from hospital not earlier than a month after the onset of the disease.

A patient recovered from Botkin's disease must for six months be kept under medical observation and suspended from arduous physical jobs. Pregnant women are subject to special registration. For 1-2 years all who have suffered from Botkin's disease are relieved from prophylactic vaccinations and when anthelmintic therapy is required the application of vermifuges (except oxygen) is forbidden because they are likely to cause a relapse of Botkin's disease by their action on the liver tissue. Special attention should be paid to the patient's diet following recovery. Frequent feedings are recommended with adequate intakes of proteins (100-200 g), restrictions on fats (50-60 g in 24 hours), and a liberal quantity of carbohydrates (450-500 g). Meat and fish are boiled or stewed. Vegetables are stewed, baked, or boiled. Beef, veal, chicken, and lean fish are preferable. The recommended dairy products are milk, kefir, yoghurt of boiled milk, skimmed cottage cheese, baked curds, as well as curd puddings, and dumplings. Not more than one egg yolk is allowed daily. Alcohol is forbidden. A strict dietary regimen should be followed for 2-3 months. If no exacerbation has been noted within 6 months the patient may be transferred to conventional diet.

Prophylaxis. In preventing the spread of Botkin's disease an important role is played by certain hygienic measures, particularly when an epidemic outbreak is present. Fruit and vegetables must be washed and treated with boiling water before they are eaten. Water must be kept boiling for 10-15 minutes. To prevent contamination of foodstuffs with the causative agent of Botkin's disease measures should be taken to destroy flies, protect foodstuffs against contamination by flies (by stretching gauze or metal nets over windows, doors, and transoms), keep courtyards clean and tidy, eradicate fly-breeding places with DDT and hexachlorane. In order to protect children, especially those who have been in contact with sick persons, the former are given intramuscular injections of 20 ml of γ-globulin which increases their resistance to the virus. The beneficial effect of these injections is due to the fact that immune bodies are produced in patients with epidemic hepatitis. These bodies are contained in γ-globulin and render the recipient immune to infection. This immunity, however, lasts not longer than 3-4 weeks. Another prophylactic measure against Botkin's disease is a comprehensive examination of blood donors suspected to have a history of this disease. Highly essential for preventing inoculation hepatitis is a thorough sterilization of instruments used for injections and other manipulations. Syringes and other instruments must be sterilized at a temperature of 120°C for 90 minutes.

ACUTE DYSTROPHY OF THE LIVER (DYSTROPHIA HEPATIS ACUTA)

Acute dystrophy of the liver is usually secondary to Botkin's disease running an unfavourable course and is rarely encountered as a primary disease. The severe affection of the liver in such cases is possibly due to the virus being especially virulent and the body resistance being undermined by antecedent diseases. Syphilis, pregnancy, and abortion predispose to a severe course of parenchymatous hepatitis and its progression to acute dystrophy of the liver. Once Botkin's disease has developed into this stage, the course of the infection abruptly deteriorates. Jaundice becomes more pronounced and signs of a severe derangement of the central nervous system make their appearance. The liver, which is slightly enlarged at the onset of the disease, begins to diminish in size and acquires a pasty consistency. A moderate enlargement of the spleen is noted frequently. The urinary secretion is reduced and its sediment contains a considerable amount of protein and leucine and tyrosine crystals.

Acute dystrophy commonly runs its course with a subnormal temperature but in case of a considerable destruction of the hepatic parenchyma the fever may rise to 39°C. The initial symptoms of languor, apathy, and somnolence are superseded by intense anxiety. There is disorientation in space, acoustic and visual hallucinations, and delirium. These are followed by convulsions, bleeding from the gums, nosebleed, extravasation of blood, and coma, the patient dying without regaining his consciousness. The above-mentioned symptoms are due to total hepatic insufficiency, known as hepatargia.

At autopsy the liver is markedly reduced in size and weighs not more than 0.5 kg; it is flaccid, atrophic, and pasty in consistency. On section yellow patches are visible (fatty degeneration), hence the obsolete name of this disease—acute yellow atrophy of the liver. At places there are foci of redness, while microscopy reveals degeneration and necrosis of hepatic cells.

Subacute dystrophy of the liver. The acute and subacute forms of dystrophy of the liver are variants of one and the same disease process. The only difference between them consists in that in subacute dystrophy decomposition of the liver tissue and intoxication fail to reach a fatal degree and are attended with reparation of the damaged liver parenchyma, owing to which the disease commonly ends in recovery. Fairly frequently, however, this disease suddenly aggravates and develops into acute dystrophy. In prognostication great caution should be displayed with regard to pregnant women and emaciated and elderly persons.

The treatment of both acute and subacute dystrophy of the liver follows the same pattern as that employed in Botkin's disease. The diet must consist of milk and vegetable foods, with plenty of carbohydrates, fruit, fruit juices, and cottage cheese. Magnesium sulphate in a dose of 15-20 g is administered daily. A liberal intake of liquid and intravenous infusions of a 40 per cent glucose solution once or twice daily in a dose of 50 ml with 10 U of insulin are recommended. A 5 per cent glucose solution in a dose of 400-500 ml should be injected subcutaneously. Compolon in intramuscular injections in doses of 2 ml every other day, and vitamins A, B, C and K are also prescribed.

WEIL-VASILYEV'S DISEASE (ICTEROHAEMORRHAGIC LEPTOSPIROSIS)

Weil-Vasilyev's disease is an acute infection due to the *Leptospira*, and is characterized by undulating fever, inflammation of the liver, jaundice, bleeding and involvement of the kidneys.

This disease was almost simultaneously described late in the 19th century (1886) by Weil and Vasilyev, and only 20 years iater its specific causative organism *Leptospira icterohaemorrhagiae* (icterus—jaundice, haemorrhage—bleeding), was discovered by Inada and Ido of Japan.

Leptospira icterohaemorrhagiae exhibits a fine spiral configuration and varies in length from 10 to 12 mm. It is often curved like a worm, thickened at the edges, and is best visible under an ultramicroscope against a dark background.

Aetiology. Weil-Vasilyev's disease occurs in epidemic form, and is spread by rats and sometimes by field-voles, shedding leptospiras in their urine. Man acquires infection through water and food as well as soil contaminated by the urine of infected rats and mice and also of sick people. The disease is encountered all year round but is more frequent in periods of an increase in rat population. A swampy terrain is a factor contributing to the spread of the disease.

The disease is world-wide in distribution but is especially prevalent in tropical and subtropical countries. Children are less susceptible to this infection than adults. The disease is not prone to relapse because it produces stable immunity.

Pathogenesis. Leptospiras gain access to the human body through the mucous membranes of the oral cavity, nose, and gastrointestinal tract, and through lesions of the skin. By the end of the first week the organisms accumulate in the liver, spleen, and lymph nodes, and by the end of the second week, in the kidneys. A number of changes characteristic of this disease occur in the infected human body. Punctate haemorrhages in the gastrocnemius muscles and a serous inflammation in the brain meninges are discovered. There is dystrophy and decomposition of hepatic cells, protein and fatty degeneration of the liver, and protein degeneration of the kidneys. The spleen is enlarged. Leptospiras are found in the liver, kidneys, adrenal glands, muscles, and nerve tissue. They are particularly numerous in the kidneys because they are eliminated through the latter within 2-3 days of the disease in great numbers.

Clinical picture. The incubation period lasts 3 to 14 days, rarely longer. The disease sets in with a sudden rise in temperature to 38-39°C, without prodromal symptoms and with a severe chill. Fever climbs to its peak on the second day of the disease and persists at a high level for 5 to 12 days. The temperature falls steeply or gradually in the morning and rises in the evening. The fever is undulating in character. The first attack of fever is followed after a 7-10 days' period of remission by another attack, less severe and shorter than the first. Such attacks of fever may recur twice and more often during the disease. In certain cases, leptospirosis runs its course without fluctuations in temperature, which persists at a level of 37-38°C as long as the disease lasts. The average duration of fever is one month. A characteristic sign of the initial febrile phase is hyperaemia of the face, redness of the vessels of the sclerae and eye conjunctiva, distinct catarrhal manifestations in the fauces, tonsils, and hard palate, herpes on the lips, the wings of the nose, and cheeks, as well as symmetrically arranged punctate haemorrhages on the chest, back, abdomen, and in the mucous membranes.

Bleeding from the nose, gastrointestinal tract, less frequently from the lungs, kidneys, and genitalia is another finding.

During the febrile phase the patient complains of nausea, vomiting, loss of appetite, a distaste for food, constipation, sometimes diarrhoea, and pains in the epigastrium and right hypochondrium.

Another cardinal symptom of Weil-Vasilyev's disease is the presence of pains in the gastrocnemius muscles, the lumbar region, and the lower extremities, as well as a high-degree general muscular weakness.

Biliary leptospirosis is attended with considerable derangements in the central nervous system. The most frequent complaints are those of stubborn headaches and insomnia. There is rigidity of the occipital muscles and, in severe cases, delirium. The cerebrospinal fluid may contain leptospiras. An important finding in Weil-Vasilyev's disease is jaundice, which makes its appearance within 4-8 days of onset and progresses until the 9th-10th day of the disease. Simultaneously one may note an increase in the bilirubin content of the blood, the presence of bilirubin and urobilin in the urine, discoloration of the stools, a uniform enlargement of the liver and sometimes of the spleen also, without substantial disorders in their function. Jaundice may sometimes be quite moderate. The blood exhibits anaemia, neutrophilic leucocytosis, aneosinophilia, and a reduction in the number of lymphocytes and thrombocytes. The ESR is considerably elevated, particularly in the febrile phase.

Involvement of the kidneys is one of the cardinal manifestations of Weil-Vasilyev's disease. The kidneys usually present a picture of focal nephritis and dystrophy of the tubules. In the urine there appears protein (up to 1-3 pro mille), erythrocytes, and hyaline and granular casts. In certain cases the urinary secretion is diminished or absent altogether. There is palpitation, a slow pulse, and a reduced arterial pressure (icteric stage). The changes in the heart are suggestive of infectious myocarditis.

In respect of its course Weil-Vasilyev's disease may be divided into the following three forms: (1) hepatorenal, when the principal symptoms are those of liver and kidney diseases; (2) meningeal, with distinct manifestations of a derangement in the central nervous system; (3) intestinal, with signs of indigestion dominating the clinical picture. This division is highly conventional since the above-mentioned abnormalities are observed in a varying degree in all patients with biliary leptospirosis.

Prophylaxis. Measures to be taken to prevent the spread of Weil-Vasilyev's disease are as follows: (1) immediate accommodation of the sick at an infectious disease hospital; (2) prohibition of the use of water from reservoirs which are known or supposed to be contaminated; (3) prohibition of bathing in water reservoirs with poor sanitary conditions; (4) the use of only boiled or chlorinated water for drinking and washing; (5) extermination of rats and field-voles in foci of infection, and disinfection of rodent habitats; (6) protection of foodstuffs against contamination by rodents.

Treatment. This consists in subcutaneous or intramuscular injections of 50-60 ml of serum obtained from persons recuperating from biliary leptospirosis. The blood of convalescents has been found to contain antigens destructive for leptospiras. Serum for injections is taken within 2-3 weeks after the abatement of fever. A similar serum may be obtained from immunized horses.

Drug therapy usually consists of administrations of penicillin in a dose of 100,000 U 5-6 times in 24 hours and streptomycin in a dose of 500,000 U twice daily until fever subsides. In relapse of temperature antibiotic therapy is resumed. Anaemia calls for transfusions of blood and blood plasma, and vitamin B_{12} injected in doses of 100-150 γ twice weekly. Vicasol is helpful in bleeding. In other respects the treatment follows the same patterns as in Botkin's disease.

TOXIC HEPATITIS

Toxic hepatitis is due to intoxication with certain drugs (novarsenol, salvarsan, atophan, chloroform, sulphonamides, aspidium), industrial poisons (phosphorus, arsenic) and poisonous mushrooms. In severe cases there is disintegration of hepatic tissue, and in mild cases, a partial fatty degeneration of hepatic cells. Disintegration of cells in toxic hepatitis is accompanied by inflammation of the interstitial tissue of the liver and the reticuloendothelial tissue.

Toxic hepatitis fairly frequently involves an affection of the kidneys and bone marrow, with suppression of the haemopoietic function.

A typical clinical example of toxic hepatitis is *salvarsan jaundice*, which occurs during the treatment of syphilis and sometimes malaria and relapsing fever with salvarsan preparations containing arsenic. The disease sets in 1-4 months after the completion of treatment and its course is in the main identical with that of Botkin's disease. In certain cases, particularly in pregnant women, emaciated and elderly persons salvarsan jaundice terminates in acute dystrophy of the liver. There are contradictory views on the causes of the disease in question. Some attribute it to the toxic effect of arsenic on the hepatic parenchyma, others, to a disease of the liver itself. According to other views, salvarsan causes the death of a considerable number of spirochaetes harboured in the liver, which fact is responsible for the tissue reaction in this organ accompanied by a rise in temperature, jaundice, etc. In certain cases there is a likelihood of Botkin's disease virus being introduced into the body when inadequately sterilized syringe and needles are used for injecting salvarsan. Nevertheless it should be presumed that salvarsan jaundice is also basically consequent upon toxic damage caused with arsenic to the liver already exposed to an earlier infection—syphilis or malaria.

In all cases providing evidence of a liver dysfunction one should refrain from the application of salvarsan, atophan, and aspidium, especially in case of pregnancy, inasmuch as the latter is fairly often attended by a liver involvement in toxaemia. Upon recovery from salvarsan jaundice, arsenic therapy may be re-instituted not earlier than after a period of 18 months. The treatment is carried out along the same lines as in Botkin's disease.

CHRONIC HEPATITIS (HEPATITIS CHRONICA)

In chronic hepatitis there is a diffuse affection of the liver with a predominant involvement of its parenchyma (parenchymatous or epithelial hepatitis) or the interstitial tissue (interstitial or mesenchymal hepatitis). The spleen also becomes involved in the disease.

Aetiology and pathogenesis. The causes of chronic hepatitis are, in the order of frequency, infections, toxic affections, and partly alimentary factors which contribute to inflammation of the liver.

Most commonly chronic hepatitis develops from an acute form caused by Botkin's disease or is a sequel to such chronic infections as malaria, syphilis, brucellosis, tuberculosis, subacute bacterial endocarditis, etc. Chronic hepatitis may be due to a chronic intoxication with lead, benzene, manganese or salvarsan, but in the Soviet Union these causes have been reduced to a negligible proportion by appropriate clinical and prophylactic measures.

Infection and toxins gain access to the liver with the blood stream via the portal vein and by way of the bile ducts from where intestinal bacteria and their decomposition products penetrate into the liver, damage the hepatic cells and intrahepatic capillaries, increasing their permeability and causing a serous oedema with atrophy of the hepatic cells.

The majority of authors believe that alcoholic intoxication is liable to cause dystrophy and atrophy of the hepatic cells to be followed by connective tissue growth. A contributing role is also played by the products of inflammation arriving from the gastrointestinal tract via the portal vein or the bile ducts, as it is fairly often seen in chronic alcoholics. Dietary deficiencies are also largely responsible for chronic hepatitis. A prolonged absence of balanced protein diet and vitamins of the B complex with an excessive fat intake may produce fatty infiltration of the liver followed by connective tissue growth.

Pathologic anatomy. In parenchymatous hepatitis the hepatic cells exhibit fatty infiltration and necrosis, while foci of newly-formed hepatic cells and a tissue response from the reticuloendothelial system of the liver occur. With progression of the disease, connective tissue proliferates, mainly about the ramifications of the portal vein, also about or inside liver lobules. At the outset of the disease the liver is enlarged uniformly and is soft in consistency, whereas in the final stages it becomes firm, granular, and reduced in size.

Clinical picture. Both the patient's complaints and the clinical manifestations, course, and complications of this disease vary with the predominant involvement of the hepatic parenchyma or mesenchyma; therefore, both forms will be described separately for the sake of convenience.

In *parenchymatous* (*epithelial*) *hepatitis*, the patient complains of debility, early fatigue on exertion, decreased appetite, dyspepsia, discomfort and pains in the right hypochondrium and epigastrium, and pruritus.

Gastric juice acidity is reduced or absent. The stomach is distended, persistent constipation alternates with brief diarrhoea sometimes associated with an intake of fatty food. The liver and spleen are enlarged.

A conspicuous sign of parenchymatous hepatitis is unmarked jaundice with retention of bile acids and dysfunction of the liver. The latter is manifested by itching and scratched lesions, urobilinuria, an impairment of the carbohydrate metabolism and prothrombin-forming function, hypocholesterolaemia, and hypoproteinaemia. Especially low is the amount of albumins with an attending increase of globulins in the blood. There is macrocytic anaemia caused by malabsorption from the intestine of vitamin B_{12}, folic acid and iron which play an important role in haemopoiesis. Anaemia is partly due to haemolysis caused by spleen enlargement, nutritional deficiency, as well as a lesion of the bone marrow. Parenchymatous hepatitis may be accompanied by leucopenia which is in part accounted for by the inhibitory effect produced on leucopoiesis by the enlarged spleen. Predisposition to bleeding is caused by a disturbance in prothrombin production. The cardiovascular system exhibits no particular changes, except for bradycardia and hypotension in jaundice. The stools are not discoloured and contain a large amount of fat and indigested muscle fibres, which is apparently associated with a derangement in the external secretion of the pancreas.

In *mesenchymal hepatitis* the patient complains of pains radiating to the back and the scapular region and a feeling of heaviness in the right hypochondrium. The pains are intense and grow much worse after a hearty meal and on distention of the intestine. The greater intensity of pains compared to those felt in parenchymatous hepatitis is accounted for by inflammatory processes which result in adhesions involving the surface of the liver and its peritoneal coverings. In diffuse mesenchymal hepatitis the liver is enlarged uniformly, whereas focal hepatitis produces an enlargement of only isolated parts of the liver which becomes lobular and nodular in appearance. The liver is firm and tender on palpation, while enlargement of the spleen is notable in diffuse affections of the hepatic mesenchyma when the reticuloendothelial tissue of the liver and the spleen become involved in the disease process. Mesenchymal hepatitis is sometimes attended with undulating fever and a chill, apparently of an infectious origin.

The function of the liver is, as a rule, undisturbed and jaundice is absent. The latter's appearance may indicate an involvement of the liver parenchyma, haemolysis of the blood (malaria), and obstruction of the bile ducts by a syphilitic gumma. Sometimes there is leucocytosis caused by the principal infection. Changes may occur in the organs involved in the principal disease process, for example, syphilitic hepatitis may be attended by mesoaortitis, tuberculous hepatitis by pulmonary tuberculosis, etc.

Course and complications. Chronic hepatitis resolves favourably if its causes have been eliminated and therapeutic measures taken in good time. It may be complicated, however, by cholecystitis, cholangitis and pancreatitis.

Differential diagnosis. The first step is to differentiate between mesenchymal and parenchymatous hepatitis. Enlargement of the liver and spleen is observed in many other diseases: circulatory disturbances (congested liver), amyloidosis, fatty degeneration of the liver, cholecystitis and angiocholitis, leukaemia, pernicious anaemia, etc. An amyloid liver is found with chronic suppurative processes in combination with an affection of the kidneys; fatty infiltration of the liver is seen with tuberculosis of the intestine, diabetes mellitus and adiposity. Diseases of the haemopoietic system are recognized, above all, from the condition of the blood.

Prophylaxis and treatment. Prophylaxis consists in measures to eliminate the aetiologic factors contributing to chronic hepatitis (Botkin's disease, leptospiral jaundice, malaria, syphilis, etc.). Particularly efficacious is an early treatment by means of adequate nourishment, a hygienic regimen and application of specific remedies against syphilitic, brucellous and malarial hepatitis. In hepatitis due to Botkin's disease appropriate therapy is required (see "Botkin's disease"). Health resort therapy is also recommended.

CIRRHOSIS OF THE LIVER

Cirrhosis of the liver is the final stage of chronic hepatitis. In this disease the liver is considerably indurated. Even at an early stage of the disease the liver has a rusty-icteric colour on section (Gr. *cirrhos*—reddish-yellow).

This disease was for the first time discovered in a Venetian senator, who was addicted to alcohol and suffered from an abdominal enlargement and indigestion. After his death autopsy revealed a considerable induration of the liver, enlargement of the spleen and a collection of fluid in the abdominal cavity (ascites). Analogous changes were discovered in 1819 by Laennec of France in a chronic alcoholic. In view of this the disease was named alcoholic cirrhosis of the liver or Laennec's cirrhosis. Later, in 1842, an association was established between cirrhosis of the liver and its chronic inflammation.

In 1875, Hanot described a case of cirrhosis of the liver which was considerably enlarged. Since then two variants of cirrhosis have been distinguished: one with shrinkage of the liver (atrophic), the other with enlargement of the liver (hypertrophic). It has been found, however, that mixed forms also exist.

Cirrhosis is widespread in Egypt, India, China and the Malayan Archipelago. The considerable frequency of this disease in a subtropical climate is associated with a fairly high incidence of malaria, yellow fever, and Botkin's disease which are factors predisposing to cirrhosis. Botkin's disease is a frequent cause of cirrhosis. The rate of cirrhosis shows a noticeable increase after an outbreak of epidemic hepatitis.

The cause of cirrhosis may be infection, inflammation, toxins, avitaminosis and alcohol. Among the noninfectious factors contributing to the development of cirrhosis alcoholic intoxication is the most important. Evidence in favour of the existence of cirrhosis of the liver due to alcoholic intoxication is provided by a large number of observations and extensive statistical data. According to Britain's state statistical reports, the death rate in cirrhosis of the liver due to chronic alcoholism is fairly high. The presence of cirrhosis is noted in 19 per cent of cases of delirium tremens. Cirrhosis is especially frequent among personnel of restaurants, distilleries, etc., which by reason of their occupation are likely to become alcohol addicts. In addition, there is evidence in favour of a direct effect produced by alcohol on the liver. This is proved by the fact that in persons who have been abusing alcohol for dozens of years, even in comparatively small quantities, a distinct picture of this severe disease of the liver is ultimately produced. Many authors assert that in chronic alcoholism the liver tissue undergoes dystrophic changes terminating in connective tissue growth, i.e., cirrhosis. At the same time quite a few facts are adduced disproving the role of alcohol in the development of cirrhosis. Firstly, it does not develop in all cases of chronic alcoholism and sometimes occurs in persons leading an extremely sober life or consuming alcohol in small doses and occasionally. In addition, a number of authors assert, not without reason, that cirrhosis is not due to a direct effect of alcohol on the liver. They believe that this disease is consequent upon the formation of toxic substances, products of decomposition and fermentation (acetic acid, skatole, indole, ammonia, etc.) and in chronic inflammatory processes occurring in the gastrointestinal tract due to chronic alcoholism. These products are absorbed from the intestine and produce a toxic effect on the liver.

Apparently some other factors exist which play a decisive role in the development of cirrhosis in persons suffering from chronic alcoholism or in those habitually consuming alcoholic beverages, even in small quantities. Possibly a certain role is played by hereditary predisposition evidence of which is often observed in practice; for example, cirrhosis of the liver was found in several children of one and the same family. A case of cirrhosis in four-year twins who had been drinking cognac daily since the age of eighteen months has been described in the literature. Undoubtedly important factors contributing to the development of this disease are chronic inflammatory processes in the gastrointestinal tract. The development of cirrhosis of the liver due to chronic alcoholism is associated with a dietary abnormality (deficit of proteins, carbohydrates, and the principal vitamins). This view is confirmed by the fact that persons suffering from chronic alcoholism consume little proteins. In addition, during inflammatory processes in the gastrointestinal tract, which are present in chronic alcoholism almost invariably, the synthesis of vitamins and their absorption in the intestine are disturbed. A dietary deficiency leads to fatty infiltration of the liver, which is a precursory of its cirrhosis. As far back as 1910 Saltykov proved the development of fatty infiltration, necrosis and proliferation of connective tissue in animals (rats) systematically given alcohol. Best (in 1949) drew a similar conclusion after inducing fatty dystrophy and cirrhosis of the liver in rats kept on a protein-free alcohol diet.

Cirrhosis of the liver may also be encountered in nonalcoholics if their diet is deficient in proteins and vitamins.

It should by no means be inferred that cirrhosis of the liver is essentially due to alimentary factors. The principal role in its development is played, above all, by acute and chronic infections, particularly Botkin's disease, syphilis, malaria, brucellosis, tuberculosis of the liver, leptospirosis of the liver, inflammatory processes in the bile ducts, and chronic intoxications of the liver with industrial poisons (lead, benzene, arsenic, copper, etc.). Alimentary factors play only a contributing role in the origination of this disease, as is the case in alcoholic cirrhosis. Cirrhosis of the liver may also develop from endocrinic and metabolic disorders (Basedow's disease, diabetes mellitus, obesity, etc.). Different forms of cirrhosis have many features in common; each of them, however, runs a specific course.

Atrophic Cirrhosis of the Liver

At the outset of the disease there is enlargement of the liver. In time connective tissue develops along the blood vessels, envelops the liver lobules and causes a severe contraction and atrophy of the liver. Because of connective tissue contraction the liver becomes markedly diminished in size, with symptoms of congestion developing in the portal system and ascites. Simultaneously numerous

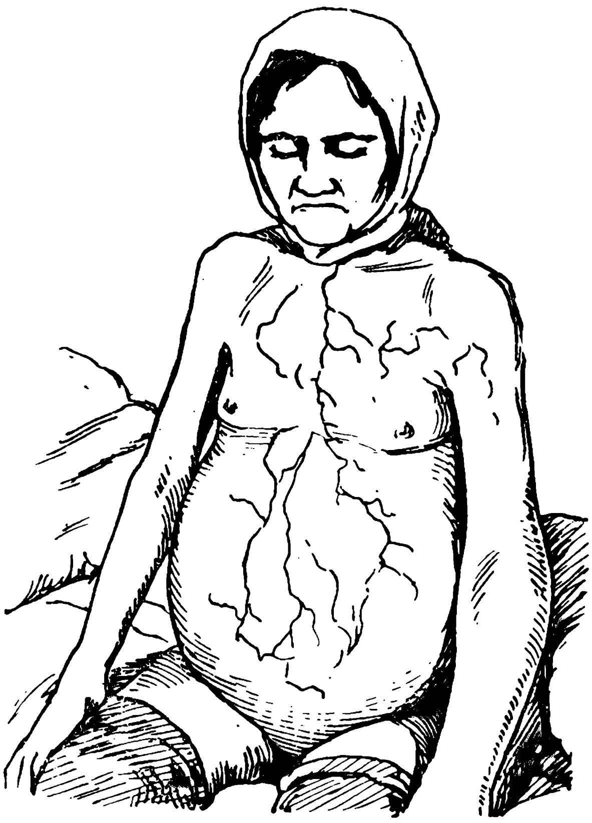


Fig. 69. A patient with cirrhosis of the liver

collaterals of the abdominal veins develop. Because of a pressure increase in the portal system (portal hypertension) this form of cirrhosis is known as portal or Laennec's atrophic cirrhosis. Many authors also call it alcoholic cirrhosis.

The clinical picture of this disease varies with its stage. In the early period when ascites is still absent the patient complains of pains about the liver radiating to the back.

The pains are caused by overfilling of the stomach and an inflammatory process in the liver. There is meteorism and distention of the intestine which are usually precedent to ascites. Portal writes in this connection that like anywhere in nature rain is preceded by wind. The patient complains of constipation alternating with diarrhoea, particularly after ingestion of fatty food, anorexia, insomnia, fatigability and lassitude. The liver is uniformly enlarged, firm and painless on palpation. There is also enlargement of the spleen. The urinary secretion is reduced. Already in this period a characteristic network of veins directed from the navel to the right hypochondrium and epigastrium is visible on the skin of the thinned abdominal wall (Fig. 69). In the lower portion of the oesophagus and the cardial part of the stomach there is a varicose dilatation of veins which produces haemorrhage, sometimes highly dangerous to life. Haemorrhoids are also encountered. In the period of ascites severe distention of the intestine is joined by a rapid or slow collection of fluid in the abdominal cavity. It is characteristic that the more the number of dilated veins in the abdominal wall, the less the amount of fluid in the abdominal cavity. In ascites the abdomen acquires a spherical form in a standing posture, and that of the frog belly, in a recumbent posture. Ascites is caused by a mechanical obstruction to the blood flow in the liver produced by scar tissue and nodules within it. A certain role is played by a reduction in the albumin content of the blood since albumins are known to retain fluid. In severe ascites the navel is protruding, and the abdominal wall is thinned and tense, sometimes covered with small whitish scars resulting from subcutaneous ruptures. Oedema occurs in the lower extremities due to a mechanical pressure from ascitic fluid on the inferior vena cava. In this period the patient has a characteristic appearance owing to a sharp contrast existing between his large belly and haggard face, a sharp nose, prominent cheek-bones, hollow cheeks and thin arms. There is a dilatation of minute vessels (telangiectasis) which resemble spiders or starlets in shape, on the neck, chin, and hands. These dilatations develop during an exacerbation of the disease. In persons with cirrhosis of the liver the palms and soles are reddened.

With progression of portal hypertension cardiovascular function is considerably impaired: there is a decrease in the mass of circulating blood because blood is collected in the so-called blood depots—the liver and spleen. The blood flow is slowed down, the arterial pressure is reduced, and tachycardia and cyanosis of the extremities appear. In this period, a further impairment of digestive function comes on the stage: the appetite is reduced or absent, the tongue is smooth, as though polished, and covered with fissures due to deficiency of vitamins of the B complex; hydrochloric acid of the gastric juice is reduced in quantity or absent. The stools contain a large amount of neutral fat and undigested muscle fibres, possibly due to a concurrent affection of the pancreas; diarrhoea is frequent.

The blood presents a picture of macrocytic anaemia, apparently associated with a deficit of vitamin B_{12} and folic acid. There is proneness to leucopenia, particularly in enlargement of the spleen, to monocytosis due to stimulation of the reticuloendothelial system, and to bleeding resulting from malabsorption of vitamin K and a reduction in the amount of prothrombin, an important factor of blood coagulation. Vitamin A deficiency occurs in consequence of insufficient conversion of carotin into vitamin A as a result of which night-blindness (nyctalopia) ensues. The amount of vitamin B is also reduced considerably.

Involvement of the nervous system are manifested by polyneuritis, a state of depression, sleepiness in the daytime and insomnia at night. Mental disorders, such as delirium, are also seen infrequently. There is a decrease in sexual desire and a disturbance in the menstrual cycle. Cachexia develops in the final stage of the disease.

The course of the disease. The disease may take a malignant course and terminate in death within several months. Most commonly, however, portal cirrhosis of the liver runs a prolonged course lasting from 10 to 20 years with periodic relapses and remissions. The most frequent complications are erysipelas, pulmonary tuberculosis, peritonitis of pneumococcic or streptococcic origin, thrombosis of the portal vein, and sometimes cancer of the liver associated with cirrhosis.

The most frequent causes of death are erysipelas, pneumonia, hepatic failure, bleeding from varicose veins, and cardiovascular failure.

The prognosis is ominous. Recovery is possible only in the initial stage of the disease when no scar tissue has yet developed. To relieve the patient's condition in considerable ascites surgeons sometimes suture the omentum to the anterior abdominal wall (Talma's operation—omentopexy) or establish an anastomosis between the portal vein and the inferior vena cava (portacaval shunt).

Postnecrotic Cirrhosis of the Liver

The development of postnecrotic cirrhosis is in most cases associated with Botkin's disease, its anicteric form including. Apparently, the transition of acute hepatitis to cirrhosis in such cases is due to a digression from the hygienic and dietary regimen either at the height of the disease or in the period of abatement of the inflammatory process. A contributing role is played by predisposing factors. in particular chronic alcoholism, etc.

Pathologic anatomy. The liver is enlarged and its colour is reddish-brown. On section reddish nodular areas are seen beside those of a yellow-green colour. Necrotic foci alternate with nodules of cellular degeneration of liver tissue. These nodules may be so massive as to lend the liver a nodular appearance. The remaining hepatic cells show signs of protein and only partly fatty infiltration. The connective tissue is moderately developed; arising about necrotized cells it grows between and inside the lobules.

Clinical picture. As distinct from portal cirrhosis the liver is enlarged. Usually it has an uneven surface and is firm in consistency. A liver enlarged for a long time and covered with small or large nodules may become smaller in the final stage of the disease and acquire a granular surface. Sometimes the degree of enlargement of the spleen is greater than that of the liver. There is severe jaundice with pruritus and scratched lesions which become worse in periods of exacerbation, while the stools are discoloured. The urine contains urobilin, bile pigments, and bile acids. Fever is commonly due to an inflammatory process occurring in the bile ducts or to exacerbation of inflammation in the liver, which is accompanied by dystrophic and necrotic processes. This form of cirrhosis, as a rule, produces no portal hypertension; the latter may be observed only late in the disease and is, in addition, inconspicuous Collaterals are absent or unmarked, gastric and haemorrhoidal haemorrhages are extremely rare occurrences. Ascites develops only in some cases, and is usually slight. Anaemia is absent. In isolated cases there is clubbing of the fingers and sometimes of the toes and turtle-back nails. The disease may take a malignant course and end in death within a few months. In other cases the course of the disease is favourable.

During the past few years malignant forms have occurred much less frequently because of the wide application of anti-inflammatory drugs and hormones.

Biliary Cirrhosis of the Liver

This form of cirrhosis was for the first time described by Hanot of France in 1875 as a hypertrophic cirrhosis of the liver with chronic jaundice. Later it was named Hanot's cirrhosis. Its development is associated with an inflammatory process in the intrahepatic bile ducts.

It is usually encountered in young men between 20 and 30 years of age. The inflammatory process in the bile canaliculi occurs due to the entry of infection with the blood stream or from the intestine. The causal agents are most commonly *E. coli*, enterococci, streptococci, and staphylococci. It is possible that *Lamblia* organisms sometimes found in the bile ducts contribute to an inflammation in the gallbladder and the bile ducts.

Pathologic anatomy. The liver is considerably enlarged, firm and darkened. Connective tissue proliferates about the bile ducts and inside the lobules. The large and small bile ducts are inflamed. The liver tissue is little involved. Portal hypertension is, as a rule, absent. Only in exceptional cases does contraction of the liver occur, and ascites arises from compression of the portal vein by connective tissue. According to experimental studies of N. Anichkov and M. Zakharyevskaya biliary cirrhosis develops as follows: upon a mass penetration of the causal organisms from the intestine into the bile canaliculi inflammatory changes occur within the latter in combination with dystrophy and necrosis of hepatic cells. This results in connective tissue proliferation in the liver and the latter's contraction.

Clinical picture. In the early period of the disease the patient complains of pains in the right hypochondrium and disturbances in digestion: anorexia, diarrhoea, etc. In certain cases signs of inflammation of the intrahepatic bile ducts and the gallbladder are indistinct.

Sometimes, the disease begins with tonsillitis, an acute inflammation of the gastrointestinal tract, and fever which later becomes undulating. As a rule, a rise in temperature coincides with another exacerbation of the disease process, and a fall, with an abatement of inflammation and a decrease in jaundice. Dilatation of veins in the abdominal wall (caput medusae), in the oesophagus and the rectum is observed very rarely. Ascites develops only late in the disease. Bleeding, if any, is due to malabsorption of vitamin K and insufficient prothrombin production rather than to a rupture in the vessels.

The cardiovascular symptoms are a slow pulse and hypotension. Clubbing of the fingers and toes and development of turtle-back nails are apparently due to malabsorption of vitamin B and calcium salts, and to infection. Biliary cirrhosis lasts for over ten years with periodic remissions and relapses.

Pigmentary Cirrhosis of the Liver or Bronze Diabetes (Haemochromatosis)

This disease results from deposition of a large amount of iron-containing pigment in the skin, particularly in the rubbing parts. in the liver, spleen, and pancreas. Hyperglycaemia and glucosuria occur in the final stages. The liver is enlarged and firm, the spleen is also enlarged. There are signs of growing insufficiency of liver function, which is mainly responsible for fatalities.

Prophylaxis and treatment of cirrhosis of the liver. Prophylaxis consists in alcoholism control. In addition, a well-balanced protein diet, adequate amount of vitamins, and a normal function of the gastrointestinal tract are necessary. The diet must also contain a sufficient amount of carbohydrates which are deposited in the liver in the form of glycogen and increase its resistance. The daily protein intake should be 100-120 g, with emphasis on lipotropic substances (cottage cheese) which prevent fatty infiltration of the liver. The intake of fats is sharply limited, and only a small quantity of butter, milk cream and sour cream is allowed. It is recommended to include the following foods into the diet: milk, kefir, yoghurt of unboiled or boiled milk, cottage cheese, bread, vegetables, fruit, thin jelly, lean meat, and boiled fresh-water fish. Piquant dishes, seasonings, pickles, and sour foods are forbidden. The intake of liquid must be restricted for fear of ascites, oedema and a circulatory failure. Very helpful are intravenous infusions of glucose, preferably without insulin, in view of a possible reduction in the sugar content of the blood. Vitamin B_{12}, B_6, B_1, a 5 per cent ascorbic acid solution in a dose of 1 ml with glucose intravenously, methionine in a dose of 0.5-1.0 g during 1-2 months, injections of choline in a dose of 30 to 100 mg during 1-2 months are also prescribed. Of the diuretics, hypothiaside, novurit, phonurit, and others may be used. For the same purpose, saline purgatives (magnesium sulphate, sodium sulphate) are also employed. Aspiration of ascitic fluid may be performed only in case of a severe distention of the abdominal wall, a considerable protrusion of the navel, and in cardiovascular insufficiency due to a high position of the diaphragm. It should be kept in mind that a large amount of protein escapes from the body together with ascitic fluid, which may lead to a rapid emaciation of the patient. Prevention of inflammatory processes in the bile ducts is the basis for prophylaxis of biliary cirrhosis of the liver. It is achieved by improving the condition of the gastrointestinal tract since inflammatory processes within the latter (colitis, enterocolitis, etc.) facilitate the entry of infection into the bile ducts. Another prophylactic measure is to improve the condition of the nasopharynx and oral cavity (control of tonsillitis, etc.), since chronic infections of these sites may also extend to the bile ducts. For prophylaxis of biliary cirrhosis it is recommended to carry out a timely and thorough treatment of chronic cholecystitis, cholangitis, and cholelithiasis, which are some of the major causes ot this disease. Systematic duodenal drainage with withdrawal of bile every 4-5 days during 2 months and an annual course of sanatorial treatment are also advisable. A good effect is produced by cholagogic remedies: Carlsbad salt or magnesium sulphate in one teaspoonful dose two times daily.

The most efficacious remedies are chemical drugs and antibiotics; sulphonamides are given in doses of 3-4 g daily during a period of 1-2 weeks at one week's interval. Three such courses of treatment are undertaken. Antibiotics are prescribed for a particular causal agent of inflammation, which is determined from a bile culture. The antibiotics commonly used are penicillin in a dose of 600,000-800,000 U per 24 hours, streptomycin in a dose of 0.5 g two times daily, and levomycetin or synthomycin in a dose of 0.5 g three times per 24 hours against *E. coli* . Chemotherapy and antibiotic treatment must be systematic and prolonged. In the event of candidiasis treatment must be interrupted and nystatin prescribed in a dose of 500,000 U three times daily.

If *Lamblia* organisms are found in the bile, treatment aimed at elimination of the parasites must be carried out. Additionally, methionine in a dose of 0.5 g three times daily (formula No. 194) and cottage cheese in daily quantities of 200-300 g are prescribed. In order to increase the secretion of bile frequent feedings and intake of a considerable quantity of liquid, preferably mineral water, are recommended if a portal circulatory disturbance (ascites, varicose dilatation of veins in the abdominal wall) is absent.

For liver dysfunction the same diet is prescribed as for Botkin's disease, for bleeding, vitamin K and calcium salts. The latter are administered intravenously or per os. Health resort therapy must be undertaken early and regularly.

INFLAMMATORY DISEASES OF THE BILE DUCTS

Among inflammatory diseases of the bile ducts the most frequent is cholecystitis, inflammation of the gallbladder, whereas inflammation of the intrahepatic bile ducts (cholangitis) ranks second, and that of the extrahepatic bile ducts, third. Cholecystitis is rarely encountered as an isolated condition. It is usually concomitant with inflammatory processes both in the intra- and extrahepatic ducts, sometimes with involvement of the liver (hepatitis). This concurrence is accounted for by a very close anatomic and physiologic association between these structures. Inflammatory changes in the gallbladder and bile ducts, however, are not identical in intensity. They prevail either in the gallbladder or in the bile ducts. In view of this, they will be described separately for the sake of convenience.

The causal agent gains entry to the bile ducts by the enterogenous, haematogenous, and lymphogenous routes (from the intestine, with the blood stream and with the lymph flow respectively).

Haematogenous (descending) route. The infection (streptococci, staphylococci, etc.) enters the bile ducts from the liver in tonsillitis, pneumonia, enteric fever, sepsis, etc. It is carried into the liver by the arterial blood or through the portal vein which receives venous blood from the abdominal cavity, including the intestine.

Enterogenous (ascending intestinal) route. By this route the causal organism, primarily *E. coli*, enters the common bile duct and then the gallbladder from the upper portion of the intestine. Sometimes, it simultaneously penetrates into the pancreatic duct; therefore, cases of cholecystitis concurrent with pancreatitis are frequent.

Lymphogenous route. This route is of secondary importance in the genesis of inflammatory processes in the bile ducts. Usually, infection gains access to the bile passages via the lymphatic channels in gastritis or appendicitis.

The presence of causal organisms—*E. coli*, streptococci, pneumococci, viruses of influenza and Botkin's disease, etc.—in the bile ducts is not enough to cause inflammation. Another prerequisite of the latter is bile congestion (biliary stasis) which contributes to the genesis of cholecystitis and cholangitis. A special role is ascribed to bile calculi in the development of cholecystitis.

In recent years a considerable decline has been noted in the incidence of inflammatory diseases of the gallbladder and bile ducts caused by enteric fever bacilli for which bile is an excellent nutrient medium. The rate of cholecystitis and cholangitis of septic origin has been reduced by highly efficacious prophylactic measures as well as a wide use of antibiotics.

The **predisposing factors** in the genesis of inflammatory processes in the bile system (ducts) are as follows: (1) bile calculi which damage the mucous membrane of the gallbladder and facilitate implantation of infection; (2) biliary stasis due to mechanical causes (close-fitting clothes, tight belts, pregnancy, meteorism, constipation, etc.); (3) disorders in bile secretion accompanied by spasms interfering with normal bile drainage. Biliary stasis may also result from a congenital or acquired bending of a duct (after operations on organs of the abdominal cavity). Another factor predisposing to an inflammation in the gallbladder and bile ducts is gastric achylia, in which the bactericidal effect of the gastric hydrochloric acid is excluded, and bacteria penetrate into the duodenum and further into the bile ducts. Enterocolitis and hepatitis also instigate diseases of the bile ducts. The great importance of neurogenous and psychogenous factors also deserves notice.

The development of cholecystitis often coincides with an intense emotional and nervous upset, which is likely to produce a spastic contraction of the bile passages followed by biliary stasis and inflammation.

Acute Cholecystitis (cholecystitis acuta)

Acute cholecystitis occurs in the following three forms: (a) catarrhal; (b) purulent; (c) gangrenous.

Pathologic anatomy. In catarrhal cholecystitis the gallbladder mucosa is hyperaemic and swollen, its epithelium is at places desquamated, and the cystic walls are infiltrated by leucocytes. The gallbladder contains a serous exudate. In purulent cholecystitis bile calculi are often found in the gallbladder, the latter's walls are intensely congested and tense, the cavity is distended with a yellow-stained purulent contents. The mucous membrane exhibits haemorrhages, areas of necrosis, small ulcerations, and tiny pustules while fibrin deposits occur on the serosal surface. Purulent cholecystitis is often caused by streptococci, pneumococci, and *E. coli* gaining access to the gallbladder. Gangrenous cholecystitis is encountered less frequently and is due to putrefactive bacteria entering the gallbladder. This form of cholecystitis produces multiple ulcerations and perforation resulting in peritonitis.

Clinical picture. The patient complains of intense pains in the right hypochondrium and in the umbilical region. They are associated with distension of the gallbladder by the accumulated fluid. inflammation in the surrounding tissues, and irritation of the peritoneum and bile passages above the site of spasm. In such cases. Shchetkin-Blumberg's symptom is marked in the right hypochondrium, but later it may extend to other regions of the abdomen.

An attack of pains is usually preceded by physical and mental overstrain, sharp movements or dietary abnormalities (fatty food. alcohol, etc.); cases are frequent, however, of pains appearing suddenly in an apparently healthy person. Pains radiate to the right shoulder-blade, right arm, sternum and lumbar region. Their intensity depends on the form of cholecystitis and the patient's reactivity (sensitivity). In the catarrhal form pain persists for several days and gradually abates. In the phlegmonous form pains are felt over a longer period. Purulent cholecystitis is sometimes attended with a chill. Dryness in the mouth, vomiting, nausea, constipation and meteorism are encountered. During an attack the face is moist with drops of cold sweat, the tongue and lips are dry, and the skin is pale. The patient continuously changes his position in order to alleviate pains. They grow much worse when he is lying on his right side. A muscular defense appears about the gallbladder. Even a slight palpation reveals severe tenderness in this region due to irritation of the peritoneum. Approximately in 40-50 per cent of cases the sclerae are slightly icteric. This is more commonly seen with gangrenous and purulent cholecystitis. Simultaneously there is a slight elevation in the bilirubin content of the blood and urobilin is present in the urine due to the effect of toxic substances on the liver.

Cholecystitis may run a benign or malignant course. The former is observed in the catarrhal form. In this case, the temperature is high already in the early days of the disease and later becomes subfebrile. Liver function is not disturbed and complications are rare. Pains usually subside early, and the temperature falls relatively quickly. The blood presents a picture of neutrophylic leucocytosis reaching levels of 15,000-18,000, with the emergence of juvenile cells and rod neutrophils and a reduction or complete disappearance of eosinophils. The red blood is unchanged.

Cholecystitis runs a severe course in its purulent and gangrenous forms. Fever is usually high. There is a characteristic repeated chill with a rise in temperature. The irritation of the peritoneum is more severe, and the disease process frequently culminates in perforation of the gallbladder and the development of biliary peritonitis.

Purulent, or phlegmonous, cholecystitis produces a severe general distress, prolonged pains and a high-grade fever with neutrophilic hyperleucocytosis (up to 20,000 and more). Because of intense pains, the patient has to assume a forced posture and lies on his back. The face becomes haggard, the forehead is moist with drops of cold sweat, the lips and tongue are dry, the face is pale, the tongue is covered with an ashen-grey coating, the abdomen muscles become rigid and hard as a board, the pulse is frequent, and the arterial pressure is low. This form is highly dangerous to life and requires emergency surgery. An even more severe course is encountered in gangrenous cholecystitis. In this form there is necrosis of the gallbladder wall and perforation into the abdominal cavity, followed by peritonitis. The pains at the onset of the attack are severe but later subside because of necrosis of the gallbladder wall. With progression of biliary peritonitis an intense meteorism develops, while the passage of gases and faeces is arrested. The patient complains of agonizing thirst which is due to frequent and intractable vomiting, and the features of his face become sharpened. There is a steep fall in the arterial pressure. The only chance of recovery is given by surgery to be followed by prolonged antibiotic treatment and chemotherapy.

Complications. Acute cholecystitis may be complicated by suppuration or perforation of the gallbladder, pancreatitis, pericholecystitis and peritonitis.

Chronic Cholecystitis (cholecystitis chronica)

Chronic cholecystitis is secondary to an acute form or develops gradually, and is commonly concurrent with cholelithiasis (gallstones).

Pathologic anatomy. The inflammation is confined to the cystic mucosa or invades the entire gallbladder, which becomes thick-walled. In certain areas, however, the wall is thinned due to atrophic changes. The mucous membrane is hyperaemic, haemorrhages and superficial ulcerations being found at places. Sometimes the gallbladder is perforated and becomes adherent to the surrounding organs. It is immured within adhesions and peritonitis develops on perforation. In certain cases, the gallbladder is contracted and contains no bile. In purulent cholecystitis it contains pus and becomes contracted and thick-walled. Sometimes, abscesses formed in the cystic wall rupture into the gallbladder or peritoneal cavity, producing fistulas. In other cases, lime is deposited in the cystic wall and lends it a bony consistency.

Clinical picture. Chronic cholecystitis develops as a sequel to untreated or abated acute cholecystitis, sometimes insidiously. In chronic cholecystitis the patient often complains of flatulence and heaviness in the abdomen, eructation, nausea, vomiting which fails to relieve his distress, a metallic and bitter taste in the mouth, constipation and, less frequently, of diarrhoea. The principal symptom is a constant dull pain in the right hypochondrium which grows worse during an attack, and radiates to the right shoulder-blade, and the right half of the neck and nape. Pains aggravate after ingestion of spicy and fatty food, after an intense emotional upset, etc. In the presence of adhesions they grow worse in jolty riding, fast walking, concussion of the body, sharp movements and when working in a stooping posture. The intensity and localization of pains may vary even in one and the same person and depend on his reactivity as well as the degree of adhesion of the gallbladder to the adjacent organs. Sometimes, the patient complains of a heart pain arising simultaneously with an attack of hypochondriac pain, which is possibly due to a disturbance in the coronary blood flow.

Chronic cholecystitis runs its course without fever; in some cases, however, the temperature may be elevated slightly and sometimes to a fairly high level. The disease is often attended with a decreased or absent gastric secretion. The pancreas is also often involved, which is accounted for by its close association with the gallbladder. In this condition the amount of diastase in the blood and urine is considerably increased.

In a period of remission a faint icteric colour may be found in the scleras and the palatal mucosa. The patient is highly excitable, irritable and suffers from insomnia. The capacity for work is considerably impaired, especially when the disease produces frequent attacks causing a severe nervous exhaustion.

With regard to its clinical course chronic cholecystitis may be latent, catarrhal-relapsing, and pyo-ulcerative. In the latent form, the patient sometimes complains of indigestion. Not infrequently, however, a mild fever is the only attending symptom. The catarrhal-relapsing form runs its course with frequent relapses following a dietary abnormality, overeating of roasted meat, smoked foods, piquant condiments, and abuse of alcohol. A marked intolerance of mutton, pork, duck and goose meat is noted. A relapse may also occur in the wake of various intestinal infections: salmonellosis, dysentery, etc. During a relapse there is a rise in temperature, neutrophilic leucocytosis and an elevated ESR.

Chronic pyo-ulcerative cholecystitis is encountered less frequently. It runs a severe course, attended by symptoms of high-degree general intoxication. The form produces periodic chills with a substantial rise in temperature. The liver is greatly enlarged, and there is tenderness about the gallbladder. The patient is emaciated. Anaemia and neutrophilic leucocytosis develop. Pyo-ulcerative cholecystitis may end in perforation of the gallbladder and biliary peritonitis. In the past this form of cholecystitis was known to have a highly unfavourable outcome. In recent years, however, the wide application of antibiotics and chemotherapy have made it possible to effect a cure even in neglected cases.

In the diagnosis of chronic cholecystitis an important role is played by palpation, duodenal drainage and cholecystography. Palpation of the abdomen reveals a marked tenderness in the right hypochondrium, about the gallbladder, and at the external margin of the right rectus abdominis muscle at the site of its intersection with the costal arch. Palpation in this region causes an especially acute pain in inspiration and often interferes with the latter (Murphy's sign). An inflammation in the gallbladder is also evidenced by pains appearing on a slight percussion of the region where it is located (Ortner's sign). In addition, in chronic cholecystitis pain is felt on pressure applied with the finger to the phrenic nerve (phrenicus-symptom) between the heads of the right sternocleidomastoid muscle. This symptom was described by Mussy. Pains also arise on pressure applied to the lower edge of the right scapula. An insignificant enlargement of the liver is determinable on palpation. Sometimes an enlarged right lobe of the liver is palpable above the inflamed gallbladder. Important diagnostic findings are furnished by duodenal drainage. B bile is considerably concentrated, turbid and contains mucus, leucocytes and epithelial cells which have undergone fatty degeneration. In chronic catarrhal cholecystitis the quantity of pathologic admixtures is scant. Bacteriologic examination (inoculation) of bile reveals the presence of one or another flora, usually *E. coli*, streptococci, staphylococci, etc. In isolated cases bile is not secreted because of a disturbance in the contractile function of the atrophied gallbladder due to the latter's contraction and considerable adhesions interfering with its evacuation. Gallbladder function is studied by x-rays with peroral or intravenous injection of a contrast medium (bilitrast, bilignost, etc.). In the presence of adhesions of the gallbladder to the surrounding organs the cystic shadow is deformed. In case of a contracted gallbladder the shadow is diminished and its intensity is reduced.

Treatment and prophylaxis of acute and chronic cholecystitis. Acute attacks of pain are relieved by means of spasmolytics—atropine (platyphylline), euphyllin and unamenable pains, with narcotics (pantopon, morphine, promedole) preferably in combination with atropine. The latter eliminates side effects due to narcotics: a delay in bile drainage into the duodenum, nausea, vomiting, constipations and urine retention. Application of heat in the form of hot-water bottles, fomentations and warm compresses to the liver and gallbladder region are also prescribed. Nitroglycerin is administered for concomitant retrosternal and cardiac pains. Bed rest is indicated after abatement of pains, and the patient is given tea, fruit water, and compote. Levomycetin, streptomycin, terramycin, tetracycline, and other abtibiotics are also used in view of an acute inflammatory process in the bile ducts.

The diet must be nutritious and chemically bland. The patient is given cottage cheese, lean veal and meat, steam-cooked cutlets, quenelles, minced meat, meat pudding, boiled chicken, lean sorts of fish (pike perch, perch, carp), and easily assimilated carbohydrate foods (sugar, honey, cereals, wheat bread, biscuits, fruit, fruit juices). The intake of liquid must not be restricted because it dilutes bile and facilitates its drainage into the intestine. The diet must also include mineral salts (common salt, potassium salts, etc.), vitamins C and K (vicasol), and vitamins of the B complex (B_1, B_2, B_c, B_{12}) which produce a favourable effect on the course of cholecystitis. Of the fatty foods it is allowed to consume daily 40-50 g of butter and 30-40 g of vegetable oils (sunflower seed oil, olive oil, maize oil). The latter have a cholagogic effect. When cholecystitis is concurrent with pancreatitis fat intake is sharply limited, and in the presence of gastric achylia only puréed food must be given. Fatty grades of meat, duck and goose meat, wild fowl, brains, liver, kidneys, smoked foods, pickles, marinades, spices, pungeant condiments, coarse cellulose foods, and alcoholic beverages (vodka, wine, beer, etc.) are forbidden. Food must be eaten frequently and in small portions to provide for a uniform drainage of bile, which prevents biliary stasis and, consequently, the development of infection.

In acute cholecystitis and relapses of chronic cholecystitis anti-inflammatory drugs are used. These are sulphonamides: sulphazole, norsulphazol, and sulphadimezin in a dose of 3-4 g per 24 hours, and antibiotics: penicillin in a dose of 800,000 U per 24 hours, streptomycin in a dose of 500,000 U given in combination with novocain and pyramidon twice in 24 hours during a period of 5-7 days. If *E. coli* are present 0.5-1.0 g of levomycetin is given three times daily during a week. Spasms are eliminated or considerably relieved with subcutaneous injections of atropine or with platyphylline and papaverine, hexonium, or a 0.5 per cent novocain solution given per os (or through a duodenal tube). Duodenal drainage is carried out once in every 3-5 days. A 50-ml dose of a 25 per cent magnesium sulphate solution is introduced through the tube and the du are drawn off during 2-3 hours. Each course of tre 8-10 lavages. This therapy should preferably be given When insertion of a tube is impossible the so-call is used. The patient is given 50 ml of a 25 per cent mag solution in the morning, after which he must lie c for two hours. In case of intense pains and diarr in patients with chronic colitis, lavage is not perfo lavage, with and without a tube, serves to evacua while the concentration of bile in the bile ducts is intervals between procedures of lavaging the gallbla is given one dessertspoonful of magnesium sulphate in the morning on an empty stomach. Peach, maiz may also be used.

Bile secretion is intensified by administrations doses of 0.25 g 2-3 times daily per os or intravenou cent solution in a dose of 10 ml, allochole, cholen (dehydrocholic acid). These preparations contain acids and produce a cholagogic effect. When the acids in the bile is decreased cholesterol precipitat and contributes to an intensified formation of cal in view, immortelle, barberries, maize stigma, birch b trefoil and other medicinal plants containing volati to increase the bile acid content.

Hormone therapy. To reduce capillary perme inhibiting inflammation, ACTH in a dose of 10 U t corticosteroids (prednisolone, prednisone) in a dos daily are administered.

Mineral waters are given to facilitate evacuati the intestine and alleviate inflammation in the bi

Physiotherapy. In acute cholecystitis or relap cholecystitis hot-water bottles are applied to the live region 2-3 times daily after dinner and supper, and a and poultice overnight. UHF therapy (15-20 sessi light, ionogalvanization, and application of paraffi mud to the liver and gallbladder region are pres disease process has subsided. Pine baths which have effect on the overexcited nervous system, particular of the bile ducts, i.e., in functional diseases, are

Therapeutic exercises are prescribed in the form gymnastics in periods of remission. They are part in dyskinesia of the bile ducts.

Health resort therapy is carried out in periods is especially effective in the presence of adhesions and ed in occlusion of the bile ducts and purulent chole tain cases mineral water treatment and pelotherap at the patient's permanent residence.

Surgery is undertaken in cases of purulent or gangrenous cholecystitis, complicated by a rupture or perforation of the gallbladder, and suppuration of the liver and bile ducts.

An operation is also indicated for patients with extensive adhesions of the gallbladder limiting its normal activity, as well as for those with frequent relapses of cholecystitis unamenable to prolonged conservative treatment.

Prophylaxis of cholecystitis consists in measures to eliminate various infections (Botkin's disease, tonsillitis, maxillary sinusitis, frontitis, dental caries, adnexitis, ovaritis, enterocolitis). Considerable importance is attached to measures against biliary stasis in the bile ducts. Among them are a regulated diet, treatment of chronic constipation, abstinence from overeating and from wearing tight clothes. Bile drainage is facilitated by physical activity: morning gymnastics, physical work, sports, promenade. The intake of foods rich in cholesterol (mutton, fatty grades of meat and fish, lard, caviar, brains, liver, etc.) must be restricted, because cholesterol contributes to the formation of bile calculi; frequent abuse of alcohol must be forbidden.

Lambliasis (Giardiasis) of the Gallbladder (cholecystitis lambliosa)

Lamblia organisms were for the first time described by D. Lambl in 1859, who had discovered them in intestinal mucus. The parasite is pear-shaped, measuring 10-30 μ in length and 5-10 μ in width. It attaches itself to the mucous membrane by its sucking disc which is found at the posterior, wider end, and is capable of fast motion by aid of its flagella. *Lamblia* organisms gain entry to the gastrointestinal tract with food, particularly with raw vegetables and water. Lambliasis is widespread in localities where no effective measures are carried out for destruction of rodents, flies and cockroaches, which all transmit *Lamblia* organisms.

Lamblia parasites may also be found in the intestine of a healthy person. Multiplying in great numbers they may injure the intestine and cause inflammation mainly of the duodenum and the small bowel. In diseases of the gallbladder they are likely to be discovered in the duodenal contents in 5-10 per cent of cases. It is believed by many that *Lamblia* organisms do not gain access here from the bile passages but are of a local origin, since cases are known when *Lamblia* organisms are present in the duodenal contents but no signs of cholecystitis or cholangitis are seen. Nevertheless, the parasites have more than once been found in bile taken from the gallbladder at operation. A number of authors assert that in cholecystitis the presence of *Lamblia* organisms in the gallbladder is not proof of their being the cause of inflammation. It should be presumed that the parasites, while attaching themselves by aid of their flagella to the mucous membrane of the gallbladder and bile ducts, cause mechanical irritation of the nerve endings embedded there. This is responsible for a spastic contraction of the entire bile system and for biliary stasis, which contributes to the origination of cholecystitis and, if the latter is already present, to its further progression. *Lamblia* organisms present in the bile passages form a kind of a vicious circle: while intensively reproducing in the exudate at the expense of products of inflammation in the gallbladder, they in their turn intensify the inflammatory process by the stasis they cause.

Clinical picture. There are asymptomatic cases of lambliasis of the bile ducts without clinical manifestations. Most commonly, however, the presence of *Lamblia* organisms, which are detected by duodenal drainage, is concurrent with clinical manifestations of cholecystitis. The patient complains of a feeling of heaviness and distension in the right hypochondrium, anorexia, a bitter taste in the mouth, nausea, less frequently vomiting, diarrhoea, debility, fatigability, a low-grade fever, at times distressing sensations in the heart region, etc. There is an icteric colour of the scleras, enlargement of the liver, tenderness of the gallbladder, hyperacidity and more often hypoacidity of gastric juice, and a spastic contraction of different portions of the intestine. The arterial pressure is reduced.

Lambliasis of the gallbladder has its characteristic features. Among them are frequent relapses, extension of the inflammatory process to the liver and the tissue about the bile ducts, and the development of cirrhosis of the liver. Certain patients exhibit a moderate anaemia, eosinophilia and an elevated ESR.

Lambliasis of the gallbladder is identified by means of duodenal drainage. Highly motile (living) and immobile (dead) *Lamblia* organisms are found in A bile and B bile. It is recommended to examine the bile while it is still warm, because the parasites are alive and are easily discovered by microscopy owing to their quick motion. In cold bile they die early, are immobile and often remain unnoticed. Lambliasis of the gallbladder should be considered only in those cases when *Lamblia* organisms are present in the bile simultaneously with other products of inflammation: mucus and leucocytes.

Prophylaxis and treatment. Prophylaxis of lambliasis consists in attention to personal hygiene, sanitary control of public catering establishments and water reservoirs, measures to exterminate rodents, flies, cockroaches, etc. An important step is hospitalization of patients with lambliasis of the gallbladder and intestine, since they may spread infection. The patient's clothes must be disinfected by immersion in a 3 per cent lysol solution.

Lamblia organisms are eliminated by administrations of osarsol (acetarsone) in doses of 0.3 g three times daily during a period of 5 days. The course of treatment is repeated three times at intervals of 5-10 days. Osarsol is contraindicated in liver dysfunction. Another efficacious drug is acrichine which was proposed by A. Baldin in 1939.

Acrichine is prescribed in doses of 0.1 g to be taken three times daily during a period of 5 days. The course of treatment is repeated three times at intervals of 10 days. The entire daily dose of acrichine (0.3 g) may be injected into the duodenum through a duodenal tube in the morning on an empty stomach. The latter method is the most efficacious, since, firstly, *Lamblia* organisms are expelled from the gallbladder and, secondly, acrichine produces a direct effect on the parasites.

When choosing between osarsol and acrichine the latter should be the drug of choice since it is more efficacious and does not produce any side effects except a slight icteric colour of the skin which later disappears without leaving a trace. The course of treatment should be followed 6 weeks later by examination of the faeces and bile (withdrawn through a duodenal tube) for the presence of *Lamblia* organisms.

In addition to the above-mentioned specific therapy it is necessary to employ the conventional methods of treatment of cholecystitis and cholangitis (dietotherapy, drug therapy, etc.) because acrichine or osarsol eliminate *Lamblia* organisms from the bile but the inflammation remains, though it is less stubborn and less liable to relapse.

Acute and Chronic Cholangitis (cholangitis acuta et chronica)

Aetiology and pathogenesis. The most frequent cause of cholangitis is cholecystitis, cholelithiasis, colitis, postoperative scars, new-growths in the gallbladder and the bile ducts, stenosis of the common bile duct or its obstruction with a stone, ascaris, or by pressure from outside, etc., as well as intoxication with lead, arsenic and other poisons. For cholangitis to develop the following factors are necessary: (a) obstruction to bile drainage and biliary stasis, and (b) infection.

The causal agents of cholangitis are *E. coli*, the virus of Botkin's disease, staphylococci, streptococci, and less frequently the bacillus of enteric fever. They gain access to the bile ducts by the ascending, descending, and lymphatic routes.

Cholangitis may be catarrhal or purulent. In catarrhal cholangitis the mucous membrane of the bile ducts is hyperaemic, sometimes ulcerated and infiltrated with leucocytes. The bile is turbid, with plenty of mucous or mucofibrinous exudate and desquamated epithelial cells.

In purulent cholangitis multiple abscesses and ulcerations form around and inside the ducts, which become stenosed due to cicatrization. The liver also contains small abscesses from which pus can be expressed. They are connected with the ducts and the liver in such cases resembles Swiss cheese. Abscesses may rupture into the thoracic and pleural cavities and into the lungs as a result of the formation of a cholo-bronchial fistula. When an abscess is located near the capsule it may rupture and become encapsulated, forming a subdiaphragmatic abscess attended by a circumscribed peritonitis. In certain cases, hepatitis terminates in cirrhosis of the liver.

Clinical picture. Patients with catarrhal cholangitis complain of dull and prolonged pains and a feeling of heaviness in the right hypochondrium. The pains radiate to the right shoulder-blade, arm and back. Other complaints are debility, fatigability, flatulence, anorexia and aversion to fatty food. Vomiting fails to relieve the patient's condition. The liver is enlarged and tender on palpation, and a moderate fever is also present. Duodenal drainage reveals mucus, leucocytes, and desquamated epithelial cells in A bile and C bile.

Purulent cholangitis, as distinct from catarrhal cholangitis, is manifested by a severe intoxication and is accompanied by a repeated rise in temperature with a chill followed by profuse perspiration. The fever may be subfebrile in old and debilitated persons because of a reduced body resistance. With a rise in temperature there is a light icteric colour of the scleras and the skin, high neutrophilic leucocytosis (15,000-20,000), aneosinophilia and an elevated ESR. Duodenal drainage yields a turbid bile containing flakes of mucus. The bile sediment consists entirely of leucocytes and epithelial cells. Bile cultures show the presence of colibacillus and *Paracolobactrum* organisms, less often streptococci, staphylococci and anaerobes. The urine contains bile pigments, urobilin, protein, and erythrocytes. The tongue is dry and covered with an ashen-grey coat. Palpation reveals muscular defense in the right hypochondrium and enlargement of the liver and sometimes of the spleen. Mussy's, Ortner's and Murphy's signs are positive. The prothrombin content of the blood is reduced. There is liver dysfunction. Cholangitis may produce a number of complications such as pancreatitis, pneumonia in the lower lobes, and dry or wet pleurisy. Pancreatitis results from extension of an inflammatory process from the bile ducts through lymphatic channels or from entry of infected bile into the pancreas, and is manifested by its characteristic clinical picture and an increase of diastase in the blood and urine.

Chronic cholangitis. In chronic cholangitis the bile ducts are dilated, ulcerated, and plugged with products of cell decomposition and fibrin. There are growths of polyps in the mucous membrane of the ducts. Inflammatory changes and connective tissue develop about the ducts and in the liver itself, a condition known as cholangiohepatitis. Multiple or isolated abscesses form in the intrahepatic ducts as well as in the liver itself and under the diaphragm, just as in acute cholangitis.

Clinical picture. Several forms of chronic cholangitis are distinguished: latent, protracted, accompanied by frequent relapses, and septic. In all of these forms the common complaint is a bitter, metallic taste in the mouth, anorexia and aversion to fatty food. The main complaint, however, is that of a constant pain in the right hypochondrium, irradiating to the right shoulder-blade, neck, and arm. The pain grows worse upon digression from a normal diet, physical and mental overstrain, as well as after infections: influenza, dysentery, pneumonia, etc. When the disease process extends to the liver the patient is distressed by a severe itching of the skin. During a relapse of catarrhal cholangitis there is a slight rise in temperature, which in purulent cholangitis becomes hectic.

The liver is enlarged, particularly its right lobe, firm and painless. The skin is of an intense yellow colour, although the bile drainage into the intestine is not arrested. Ortner's, Mussy's and Murphy's signs are positive. The liver function is deranged, which is established by functional tests (Quick-Pytel's, colloidal gold, thymol turbidity, corrosive sublimate, etc.), as well as from the presence of bilirubinaemia and urobilinuria. Cholangitis running a protracted course with relapses produces adhesions to the surrounding organs: the duodenum (periduodenitis), the gallbladder (pericholecystitis) and the stomach (perigastritis). The septic form of chronic cholangitis resembles subacute bacterial endocarditis in regard of its course. It is encountered extremely rarely, mainly in debilitated and emaciated persons, and runs its course with a hectic fever, embolism, anaemia, leucopenia, an elevated ESR, focal nephritis, clubbed fingers, enlargement of the liver and spleen, and severe emaciation. The patient is slightly icteric. C bile cultures reveal the presence of *Streptococcus viridans.*

Chronic cholangitis runs a protracted course; its catarrhal form is benign, while the purulent and septic forms are severe. The prognosis becomes ominous when symptoms of hepatic insufficiency appear.

Treatment. The patient is prescribed complete bed rest and is maintained on a mechanically and chemically bland diet (puréed or steamed meat, chicken, semolina and oatmeals, yesterday s wheat bread, fruit juices, thin jellies, baked apples, biscuits, etc.). Pains are soothed with applications of hot-water bottles, compresses, and poultices (if no irritation of the peritoneum is present), and administration of spasmolytics (atropine, platyphylline, belladonna with papaverine, anaesthesin, etc.). Anti-inflammatory drugs are also prescribed. These are penicillin (up to 1,000,000 U per 24 hours), streptomycin (up to 1 g per 24 hours), terramycin, tetracycline, mycerin, levomycetin and synthomycin. Usually unmarked inflammatory symptoms subside within 5-7 days; treatment, however, must be continued after an interval of 5 days. Purulent and septic cholangitis require a longer course of therapy. Patients with cholangitis must be kept under observation for a long period in view of the risks of complications requiring surgical interference.

DYSKINESIA OF THE BILE DUCTS (BILIARY DYSKINESIA)

Normal supply of bile into the intestine is ensured by contraction of the gallbladder and simultaneous opening of the sphincter situated in the neck of the gallbladder and another sphincter at the outlet from the bile duct. A disturbance in such synchronous work of the entire bile-secreting system produces dyskinesia of the bile ducts. In hyperstimulation of the vagus nerve the sphincters, instead of opening, close still firmer, and the gallbladder contraction fails to perform its function, i.e., is not completed by evacuation of bile. In hyperstimulation of the sympathetic nerve the muscular coat of the gallbladder is relaxed and the bile becomes congested within the latter. The relaxed gallbladder walls lose their contractility. Thus, a disturbance in the normal regulatory function of the vegetative nervous system produces two types of stasis in the gallbladder and the bile ducts: spastic, in hyperstimulation of the vagus nerve, and atonic, in stimulation of the sympathetic nerve. Both these variants of biliary dyskinesia develop as a result of general neurosis and are, as a rule, concurrent with other neurogenous diseases — gastric and duodenal ulcer, hypertensive vascular disease, etc.

A rare intake of food contributes to the development of this disease because in this case the flow of bile from the liver and the gallbladder is disturbed. Protracted dyskinesia may become responsible for the development of inflammatory processes both in the gallbladder and the bile ducts. These secondary inflammatory changes may culminate in chronic hepatitis and cirrhosis of the liver.

X-ray examination plays a decisive role in the diagnosis of spastic and atonic forms of biliary dyskinesia. In the latter form, the gallbladder is, as a rule, distended by bile for a long time and sometimes to a considerable size, overhanging the shadow of the liver and failing to be evacuated in some cases even after 24 hours.

Prophylaxis and treatment. Prophylaxis consists in measures to strengthen the nervous system, regulation of diet, and frequent intakes of food. Other very important measures are gymnastics, daily walks and light physical work. As distinct from cholelithiasis and cholecystitis there is no need for a strict diet. On the contrary, very useful are foods which intensify the evacuation of the gallbladder (meat, fats, proteins, sour cream, etc.). They may be given if cholecystitis is absent. In hypertonic forms of dyskinesia the diet should exclude extractive substances, poorly digestible fats, and foods rich in cholesterol (brains, kidneys, liver, meat of fat grades, spicy foods, condiments, baked foods of rich dough, etc.). Very helpful are spasmolytics: atropine, platyphylline, papaverine, euphyllin, as well as sedatives (bromides), soporifics, and physiotherapeutic procedures, including thermal, pine and saline-pine baths. Other remedies are a galvanic collar, iontophoresis with novocain in the region of the liver, and diathermy.

Health resort treatment is mainly undertaken to strengthen the nervous system. This improves the contractility of the gallbladder in atonic states, while in hypertonic forms there is a diminution or disappearance of the spasm of the ductal sphincter.

CHOLELITHIASIS

Cholelithiasis is a frequent human disease. It is particularly widespread in the United States and Western Europe.

Bile calculi (gall-stones) are contained in the gallbladder in 10-15 per cent of the population. They have been discovered even in the newborn; until the age of 15-20, however, their occurrence is extremely rare. After the age of 70 bile calculi may be found in every third person. Women are much more liable to this disease than men. The first signs of cholelithiasis usually appear at the age of 30-50 years.

Aetiology and pathogenesis. One of the most important causes of cholelithiasis is infection, which fact was for the first time noted by S. Botkin. A contributing role is played by inflammations of the gallbladder caused by enteric fever, acute and chronic colitis and enterocolitis, appendicitis, and other infections when bacterial flora gains access to the gallbladder by the ascending route. Penetration of infective germs into the bile ducts, however, is not by itself a decisive factor in the formation of bile calculi. Other conditions favouring this are also necessary, in particular stasis in the gallbladder. Stasis develops as a result of wearing tight clothes, in constipation, meteorism, as well as during pregnancy. In the latter case there is a considerable rise in the intra-abdominal pressure due to enlargement of the uterus interfering with normal evacuation of the gallbladder. Stasis in the gallbladder sometimes results from inflammatory processes in the adjacent organs, dyskinesia of the bile ducts, in connection with adhesions to the stomach, liver, and duodenum, and from other causes.

An important role in the development of cholelithiasis is played by excessive intakes of foods rich in cholesterol and cholesterol-like substances (fatty meat and fish, lard, fats, caviar, eggs, etc.). It has been noted that where the diet mainly consists of rice or vegetable foods, the formation of bile calculi is a rare occurrence.

There are many examples to prove the contributing role of cholesterol in the development of cholelithiasis. For instance, the French clinician Chauffard, the author of the theory of cholesterol origin of cholelithiasis, describes the case history of a young girl with pulmonary tuberculosis who on doctors' advice ate 11 eggs daily during a period of 3 months. This resulted in repeated and rather intense attacks of cholelithiasis. Of course, it is not only an increased level of cholesterol in the blood that matters. An important part as played by a disturbance in the balance between the content of bile acids and cholesterol of the bile. Under normal conditions this correlation is 15 : 1 whereas in patients with cholelithiasis the ratio is 6 : 1. With a considerable reduction in the amount of bile acids cholesterol precipitates from the bile.

It should be noted that hereditary predisposition also plays a certain role in the development of cholelithiasis. This disease is fairly frequent in children whose parents have various metabolic disorders: obesity, diabetes mellitus, etc. In certain cases, this is accounted for by identical dietary patterns—intakes of excessive and fatty foods, but a hereditary predisposition is also responsible.

The mechanism of stone formation and structure of stones. Bile calculi vary from a grain of sand to a chicken egg in size and from one to several thousand in number. Large stones may be isolated. Cystic stones are elliptical, liver stones, branch-like, while stones of the common bile duct are elongated. In general, all bile calculi take the shape of the site of their origination. Stones may be faceted if they are soft in consistency and suffer pressure from other stones.

Inflammatory stones contain cholesterol and calcium bilirubin and develop as a result of inflammation of the gallbladder. Desquamated epithelial cells of the gallbladder mucosa form the stone nucleus on which bilirubin, cholesterol and calcium salts are deposited. The mechanism of formation of metabolic stones is different. Stones incorporating 96-98 per cent of cholesterol are formed in case of a bile acid deficit in the bile, which leads to precipitation of cholesterol from the latter. Cholesterol crystals deposited one upon another form cholesterol stones. In addition to liver bile, other sources of cholesterol stones may be epithelial cells decomposed in inflammation of the gallbladder and elements of inflammatory products, erythrocytes and leucocytes in particular. Isolated cholesterol stones range from a pea to a pigeon egg in size. They have a thin protein layer as their basis, and are round or oval in shape, radial in structure, float in water, and give out a bright flame when burning. Their surface is smooth or wart-like. Often, with an outbreak of infection in the gallbladder bilirubin and calcium are deposited on the surface of cholesterol stones, so that mixed stones are produced. These stones are usually multiple and rather variable in shape: oval, round, polygonal and faceted. Their colour is brown or green, and their structure is laminar. As distinct from cholesterol stones they sink in water and fail to burn. Originating in the gallbladder, they may gain access to the duct, and sometimes tightly pack the gallbladder.

Pigment (bilirubin) stones consisting of bilirubin and lime, are multiple and small in size, round or branch-like, thorny, and of a black colour with a greenish hue. They are lodged in the intrahepatic bile ducts, less frequently in the gallbladder, and are so soft as to be crushed with the fingers. Usually pigment stones are found in an intensified destruction of erythrocytes, i.e., in haemolysis.

Clinical picture. Cholelithiasis manifests itself by nausea, a feeling of heaviness in the right hypochondrium, dull pains, and a feeling of pressure and fullness in the abdomen after abundant and fatty meals. In this period the liver is slightly enlarged, and the region of the gallbladder is tender on palpation. Cholelithiasis produces a characteristic biliary colic. The latter sometimes occurs in the best of health, but is more commonly preceded by gastric and intestinal dyspepsia. As a rule, it seizes the patient at night or 2-3 hours after a heavy meal when the bile begins to flow into the duodenum. Sometimes a colic ceases within several minutes or hours, but it may also last for 2-3 days, alternately receding and relapsing. Pains in biliary colic are acute and stabbing, being localized to the right hypochondrium and extending throughout the abdomen, radiating to the right shoulder-blade, right arm, the interscapular space, and less frequently to the lumbar region and left shoulder-blade. Pains may be so intense as to respond only to narcotics. Other precipitating causes, in addition to abundant and fatty food, especially at supper, are alcohol, jolty riding, chilling, great physical overstrain, mental and emotional upset, pregnancy, and the menstrual or premenstrual periods.

The pain is caused by spasm of the smooth muscles of the gallbladder and bile ducts, including the common bile duct. This spasm arises from stimulation of the mucous membrane by a stone passing through the neck of the gallbladder and the common bile duct. Most probably stones stimulate the nerve endings embedded in the mucous membrane of the bile ducts and cause a reflex contraction of the smooth musculature. In addition, a certain role is played also by a rise in pressure within the gallbladder, and an inflammation of its coats. It is worthy of note that pains are caused not only by stones and sand but also by pus, mucus and blood passing through the duct. The intensity of an attack of pain in biliary colic varies with the reactivity of the body and sensitivity of the nervous system. Pain becomes particularly acute in case of a sudden obstruction of the common bile duct. In a gradual progression of obstruction as a result of congestion in the duct walls, with a slow rise in pressure, pains are less severe and are dull in character. An attack is attended with hiccough, nausea and vomiting of bile which fails to relieve the patient's condition. Other symptoms are a bitter taste in the mouth, meteorism, and a rise in temperature, sometimes to a high level, at times preceded by a chill. Vomiting is caused by distension of the gallbladder. The rise in temperature is due to absorption of inflammatory products. It should be borne in mind, however, that the temperature may rise also in the absence of infection, after nervous overstrain, and lasts, as a rule, throughout the attack.

Biliary colics may be accompanied by cardiac pains which cause a reflex spasm of the coronary vessels and sometimes myocardial infarction. The latter is seen especially often in coronary atherosclerosis. An irregular rhythm of the heart—cardiac arrhythmia, and retention of urine followed by its abundant secretion after the end of a colic also occur.

After an attack many patients complain of dull pains in the right hypochondrium, distension of the abdomen, eructation and nausea. The liver is enlarged, the region of the gallbladder is slightly tender on palpation. Sometimes an icteric colour of the scleras, the mucous membranes of the soft palate and even of the skin appears on the next day. A more prolonged jaundice occurs in obstruction of the common bile duct. In such cases stools become discoloured and drainage reveals no bile in the duodenal contents. Patients with cholelithiasis more commonly show the absence or reduction of free hydrochloric acid of the gastric juice, and in rare cases hyperacidity.

Complications. The following complications are encountered in cholelithiasis: obstruction of the neck of the gallbladder or the common bile duct with a stone, formation of internal or external fistulas, cholangitis and cholecystitis. In obstruction of the cystic duct or the neck of the gallbladder the latter is distended and is palpable as an elastic pear-shaped body. A considerable enlargement of the gallbladder is observed when a stone is acting as a valve letting bile into the gallbladder and blocking its outflow.

The gallbladder is sometimes filled with a pale mucous fluid (up to 0.5 l). This is known as dropsy of the gallbladder. This condition may pass spontaneously or last for years, at times becoming suppurated as a result of infection of the bile (purulent inflammation of the gallbladder) with a likely necrosis, perforation and peritonitis.

Obstruction of the common bile duct with stones from the gallbladder or liver ducts causes obstructive jaundice. The latter however, may also be seen with spasm of the duct walls occurring in the presence of a small stone. Jaundice may sometimes be intermittent, if a moving stone is acting like a valve, jaundice developing upon obstruction and diminishing when the bile flow is restored after the stone passes through the narrow place. Total obstruction of the common bile duct occurs very rarely since its lumen becomes considerably distended and a passageway is formed between the duct wall and stone. Total obstruction is due to a concurrent inflammatory process as well as a spasm.

In obstructive jaundice bile is congested above the site of obstruction, overfills the bile ducts, breaks into the lymphatic channels and enters the blood stream. The amount of bilirubin, bile acids, and choleseterol in the blood is increased. The urine contains many bile pigments, while stools are discoloured, malodorous, and contain a large amount of neutral fats and bile acid crystals. A colourless juice is withdrawn on drainage. The patient complains of pruritus. There is hypotension and bradycardia. The function of the liver is deranged, and the gallbladder is not palpable because it is contracted, firmly packed with stones, and is unable to distend. Enlargement of the gallbladder in obstructive jaundice is observed in compression of the common bile duct from outside, in particular in cancer of the head of the pancreas, since in such cases the gallbladder is normal and retains its elasticity. Obstruction of the common bile duct may cause an inflammatory process in the bile passages since biliary stasis contributes to the reproduction of bacteria. Therefore, cholelithiasis is frequently accompanied by cholecystitis and cholangitis running their course with an undulating fever and chills. Pains become nagging, and a rise in temperature is attended with chills, the liver is enlarged, tender on palpation, and its function is impaired.

Obstruction of the common bile duct sometimes terminates in ulceration of the duct wall due to its compression with a stone (decubitus, necrosis) and formation of fistulas leading into the adjacent organs. Stones may be expelled with vomit or faeces. This occurs after the formation of a fistula between the gallbladder, the common bile duct, and the transverse colon. Infrequently these stones are impacted in the intestine, commonly at the site of the valve of Bauhin, causing intestinal obstruction followed by a peritoneal inflammation. Prolonged obstruction of the gallbladder culminates in disturbance of liver function, emaciation, anaemia, tendency to bleeding, and death in a comatose state.

Prophylaxis and treatment. It is necessary to eliminate all factors causing or predisposing to cholelithiasis. For this purpose frequent feedings in small portions are recommended, mainly milk products and vegetables (flour foods, sugar, vegetables, milk, kefir, cottage cheese, vitamins, etc.), avoiding food rich in cholesterol and cholesterol-like substances. Intakes of protein foods must be restricted only when the function of the liver is disturbed.

Spicy foods (sauces, condiments) and products of rich dough are forbidden. One is recommended to drink liquid in quantity since the latter dilutes bile and increases its secretion. Certain mineral waters are also efficacious. They are drunk warm 2-3 times daily. Health resort therapy is also helpful.

During an attack of cholelithiasis the patient is given subcutaneous injections of morphine or pantopon in combination with atropine, sometimes only atropine or platyphylline, which relieves spasm of the gallbladder and duct.

Surgical interference is recommended only in frequent attacks when all other therapeutic measures are ineffective. Surgery is absolutely indicated in the following cases: an imminent danger of perforation of the gallbladder, peritonitis, purulent cholecystitis and obstruction of the common bile duct for more than 2 weeks.

Relative indications for surgery are adhesions of the gallbladder to the surrounding organs (pericholecystitis), failure of a prolonged medical therapy, progressive derangement of liver function, and pancreatitis.

ECHINOCOCCOSIS OR HYDATID DISEASE OF THE LIVER (ECHINOCOCCUS HEPATIS)

This disease is due to the larval stage of a tapeworm parasitizing in the intestine of the dog, fox and wolf. Man is an intermediate host. He becomes infected when the parasite enters the intestine where it sheds its capsule. Boring through the intestinal wall the parasites enter the blood stream and are carried to various organs. However, because of the characteristic system of the portal vein, which collects blood from the intestine, they usually gain access to the liver where the larval stage develops.

Children playing with dogs are more liable to contract the infection but it may also spread by an indirect route: through water, foodstuffs and household utensils.

Having settled in the liver, the cyst grows slowly and may give no symptoms for many years. The hydatid cyst consists of two layers and is filled with a clear amber-coloured fluid. Inside the cyst, daughter cysts develop. Such echinococcus is known as a single-chamber cyst. Around the cyst there forms a connective tissue membrane which, however, fails to disrupt interchange between the parasite and host completely. Less frequently, daughter cysts grow outwards, producing a multichamber echinococcus.

The growth of the cyst causes enlargement of the liver, which is determined by palpation and percussion. No disturbance in the function of the liver is noted. An early symptom of the disease is eosinophilia which, however, is not a very constant finding and is absent in suppuration of the hydatid cyst.

The diagnosis of echinococcosis is largely aided by Casoni's test which consists in an intracutaneous injection of 0.1 ml of echinococcus antigen (hydatid fluid). If the parasite is present in the body, a white papule appears at the site of fluid injection (positive Casoni's test). The reaction occurs in 15 minutes or in 2-4 hours. In the former case it lasts 1-2 hours; in the latter, about 24 hours.

With a multichamber hydatid cyst, the liver is firm and nodular, and at later stages of the disease there is also enlargement of the spleen. The cyst exerting pressure on the surrounding organs may cause jaundice, ascites, and difficulty in breathing.

Among the complications and outcomes of echinococcosis of the liver the most serious is suppuration of the hydatid cyst with a typical picture of a liver abscess. A suppurated cyst may rupture into the adjacent organs: the lungs, the pleural cavity, and the peritoneum.

The most favourable outcome of hydatid disease is the death of the parasite followed by its calcification. More frequently, however, the prognosis is ominous, particularly with a multichamber echinococcus.

CARCINOMA OF THE LIVER (CARCINOMA HEPATIS)

Primary hepatic carcinoma is encountered rarely. It afflicts elderly persons, mostly men. An important role in the development of this disease is played by precursory cirrhotic changes in the liver, cholelithiasis, and sometimes injuries to the liver.

The following forms of carcinoma of the liver are distinguished: (a) massive carcinoma which invades almost the entire liver, the latter's centre showing a whitish mass with a softening in the middle; (b) nodular carcinoma—a tumour of a yellow colour with many daughter newgrowths; (c) diffuse carcinoma—a tumour of a yellowish-white colour involving the entire liver. Carcinoma of the liver produces metastases to lymph nodes, the lungs, less frequently to bones, the adrenals, and the intestine.

The principal complaints are anorexia, aversion to meat and rich food, vomiting, meteorism, a moderate fever, apparently caused by decomposition of the tumour, emaciation, and a growing debility.

Of the objective symptoms the most important is a considerable enlargement of the liver which grows to 4 kg and more in weight. On palpation the liver is firm, painless, its lower edge is rounded, and sometimes reaches the navel. In massive carcinoma its surface is smooth; in nodular carcinoma isolated nodules deforming the liver may be palpable on its anterior surface. Pressure from cancerous nodules on the bile ducts may give rise to jaundice, and on the portal vein, to ascites. Progressive anaemia and leucocytosis occur. The spleen is not enlarged. Liver function is at first changed but little or completely retained if the disease process is focal in character and the unaffected part performs a compensatory function. At later stages, symptoms of hepatic insufficiency develop, such as hepatic coma or haemorrhagic diathesis.

In the diagnosis of primary carcinoma of the liver great importance is attached to: (a) needle biopsy of the liver and histological examination of the punctate in which large atypical cells with blue nucleoli in the nuclei are found; (b) x-ray examination of the liver contours; (c) pneumoperitoneum and peritoneoscopy.

An early diagnosis of primary carcinoma of the liver is very difficult, since similar symptoms are seen in many diseases of the digestive tract. Identification is also difficult at later stages, particularly in diffuse carcinoma when the latter produces a clinical picture resembling those of cirrhosis and echinococcosis of the liver, and liver congestion.

The patient dies within 6-8 months of the onset of the disease.

Prophylaxis consists in a timely elimination of factors responsible for cirrhosis of the liver, cholelithiasis, etc.

The treatment is symptomatic, and in rare cases surgical.

Metastatic carcinoma of the liver is much more frequent than primary carcinoma. Carcinomas of the stomach, the pancreas, the uterus, and the ovaries often metastasize to the liver, in which nodules of variable size develop, covering the entire liver with whitish formations. Metastatic carcinoma of the liver often causes ascites as a sequel to cancerous peritonitis and involves the lymph nodes of the hepatic porta. Identification of metastatic carcinoma is not difficult in the presence of primary carcinoma in the above-mentioned organs. The nodular form which is usually secondary in origin is the easiest for diagnosis. Unfortunately, diagnostic errors are frequent, primary carcinoma of the liver being mistaken for a secondary carcinoma, and vice versa.

The treatment is symptomatic.

CARCINOMA OF THE BILE DUCTS (CARCINOMA DUCTUS CHOLEDOCHI, DUCTUS HEPATICI)

Cancer of the bile ducts is a rare disease producing a small tumour usually growing from the common bile duct, either from its initial portion, or from the site of its junction with the duodenum. The tumour early obstructs the common bile duct and causes persistent obstructive jaundice, without a precedent biliary colic, and marked by acholic stools, bilirubinaemia, cholangitis, pruritus, haemorrhages, cholaemia, etc. In addition to jaundice, there is distention of the gallbladder (positive Courvoisier's sign). Compression of the hepatic porta by a tumour gives rise to ascites. Jaundice may temporarily recede owing to decomposition of the tumour.

Differential diagnosis must be carried out between cholelithiasis and carcinoma of the head of the pancreas. In cancer of the bile ducts jaundice appears, as distinct from cholelithiasis, without a precursory biliary colic and fever. In addition, the presence of a positive Courvoisier's sign rules out the possibility of cholelithiasis. In carcinoma of the head of the pancreas jaundice is persistent and Courvoisier's sign is positive.

A patient with cancer of the bile ducts is rapidly emaciated and dies of cholaemia within 3-6 months of the onset of jaundice.

Treatment. A malignant tumour in the bile ducts may be excised surgically.

CARCINOMA OF THE GALLBLADDER (CARCINOMA VESICAE FELLEAE)

Carcinoma of the gallbladder is encountered rarely. In respect of its incidence it ranks sixth among newgrowths of the digestive tract and mainly occurs in elderly women. The predisposing factors are cholelithiasis and cholecystitis. The malignant tumour is localized to the neck and fundus of the gallbladder, extends to the liver and involves the lymph nodes at the gallbladder neck. The tumour is usually a scirrhus or adenocarcinoma.

Carcinoma of the gallbladder may go unrecognized for a long time, and manifest itself only in the late stages. The most frequent symptoms are pain, enlargement of the liver, a palpable tumour of the gallbladder, jaundice, dyspepsia (nausea, vomiting, anorexia), tenderness in the region of the gallbladder, and ascites.

Pain is felt as a rule in the right hypochondrium or the epigastrium, sometimes becomes colicky, and in certain cases radiates to the right shoulder. Similar pains occur in cholecystitis and cholelithiasis. Actually, in more than 70 per cent of cases, carcinoma of the gallbladder is concurrent with either of these conditions; therefore its early clinical symptoms are always similar to those of cholecystitis and cholelithiasis. Later, pains become constant and dull, jaundice appearing sometimes as a result of obstruction of the common bile duct or compression of the hepatic porta by metastases. As distinct from jaundice resulting from obstruction of the common bile duct with a stone, jaundice in carcinoma of the gallbladder develops without a precursory biliary colic and fever. The haemopoietic system, unlike its condition in tumours of other organs of the digestive tract, is not substantially damaged, and anaemia is, therefore, very rare. Carcinoma of the gallbladder entails no noticeable weight loss, which is possibly accounted for by a precedent disturbance in fat metabolism and the patient's death at a relatively early date.

Duodenal drainage often fails to yield B bile; x-rays reveal a delay in the passage of barium into the duodenum or a filling defect in the latter due to pressure from a tumour. Cholecystography provides evidence that the gallbladder does not function. Another diagnostic sign is an irregular fever apparently caused by decomposition of the tumour.

In view of the close connection existing between cholelithiasis and carcinoma of the gallbladder, surgery should be undertaken whenever the former disease changes its course and shows a trend to malignancy.

Death results from extension of tumour decomposition, and perforation of the gallbladder followed by peritonitis.

Diseases of the Pancreas

The pancreas performs an important function in digestion: it secretes trypsin, diastase, maltase, lactase and lipase into the intestine. Trypsin catalyzes the breaking down of proteins and their decomposition products (albumose, peptones). Diastase breaks down starch and other polysaccharides, converting them, ultimately, into glucose. Lipase splits alimentary fats into fatty acids and glycerol. Hydrochloric acid is a physiologic stimulant of the external secretory function of the pancreas.

Total failure of the fermentative function of the pancreas leads to disturbances in digestion and absorption of fats, proteins, and carbohydrates. Pancreatic achylia manifests itself by diarrhoea which emaciates the patient. The stools in this condition are loose, frequent, grey-coloured and malodorous. The faeces are rich in neutral fats and become coated with an oily film on cooling (steatorrhoea). Microscopy of the faeces reveals numerous drops of neutral fat. Sometimes there is a disturbance in protein digestion manifested by creatorrhoea. At the same time, a large number of muscle fibres with retained striae and, what is most important, nuclei is seen under the microscope. With a partial disturbance in the fermentative function of the pancreas and with conventional diet steatorrhoea and creatorrhoea are absent. The latter appear only after a test diet consisting of 50 g of butter, 100 g of bread, and 2 eggs or underroasted meat scrapings or calf's thymus rich in nuclei.

To reveal latent insufficiency of pancreatic function a diagnostic test is used: 30 ml of a 0.5 per cent hydrochloric acid solution is introduced into the duodenum through a thin tube. Normally, 30 to 60 ml of secretion is discharged within 10-15 minutes. A disorder in the external secretory function of the pancreas causes pains while the amount of secretion is extremely meager.

ACUTE PANCREATITIS (PANCREATITIS ACUTA)

Acute pancreatitis runs its course with symptoms of shock and oedema or haemorrhagic necrosis of the pancreas.

Aetiology and pathogenesis. Acute pancreatitis results from infection of the pancreas in cholecystitis, cholelithiasis, gastric and duodenal ulcer, as well as in epidemic parotitis, chronic tonsillitis, various fevers, pneumonia, malaria, etc. (haematogenous infection).

In inflammation of the pancreas the bile and intestinal juice gain access to the gland and activate trypsinogen, converting the latter into trypsin which causes necrosis of the pancreatic parenchyma. This results in autodigestion of the pancreas, particularly its adipose tissue. The disease is encountered mainly in men prone to overeating and abuse of alcohol.

Pathologic anatomy. Pancreatitis may be catarrhal, purulent, and haemorrhagic. In catarrhal pancreatitis, leucocytic infiltration is observed in the ducts, vessel walls and interstitial tissue of the gland. Purulent pancreatitis produces a diffuse suppuration and multiple small abscesses, particularly in the head of the pancreas. In haemorrhagic pancreatitis the pancreas is enlarged and succulent, with multiple sites of bleeding and necrosis, in particular fatty necrosis seen as tiny stearin patches scattered both in the pancreas and in the surrounding tissues (omentum, peritoneum, pleura, etc.). The abdominal cavity contains a haemorrhagic, less often serous, exudate.

Clinical picture. The disease begins suddenly after an abundant meal, when the digestive process is the most intense and produces epigastrial and periumbilical pains which acquire the character of girdle pains. Pains sometimes radiate to the left shoulder and sigmoid colon. Shock is encountered fairly often, the patient's face becoming ashen-grey and the pulse thready. This condition is caused by involvement of the thick nervous network of the gland in the inflammatory process and compression of the solar plexus by the enlarged pancreas, resulting in agonizing pains. During an attack flatulence, nausea, vomiting, and at times intestinal obstruction occur. No marked rigidity of the abdominal muscles is encountered.

The blood shows erythrocytosis (due to haemoconcentration), elevated leucocytosis, and an increased amount of diastase and lipase both in the blood and urine in the early days of the disease. In severe cases pains continue for hours and fail to respond even to narcotics. The presence of a high-grade fever with a chill and marked leucocytosis may suggest purulent pancreatitis. Rupture of pus into the abdominal cavity causes peritonitis. Compression of the common bile duct by the inflamed and succulent pancreas causes obstructive jaundice. In severe cases death may occur within 24 hours.

Pancreatitis may run a milder course. In such cases the patient is only discomforted by epigastrial pains or there is an insignificant increase in the concentration of enzymes in the blood and urine.

Differential diagnosis must be established between myocardial infarction, perforating gastric and duodenal ulcer, acute cholecystitis, and perforation of the vermiform appendix.

The prognosis is always ominous and in severe cases permanent observation by a surgeon is necessary.

Prophylaxis and treatment. Prophylaxis consists in measures against obesity, overeating, and abuse of alcohol. Treatment and prevention of cholecystitis and cholelithiasis are also necessary.

In severe cases measures against shock have to be taken by administration of narcotics, novocain blocking, intravenous drip of physiologic solution, blood transfusion and atropinization. Evacuation of gastric juice is recommended, and the inflammatory process is treated with antibiotics: streptomycin, biomycin, and others. Sometimes, the pancreas has to be excised or operative drainage of its affected part is performed. Fasting for 2-4 days is necessary, which is followed by meals of easily digestible carbohydrate food (sugar, white rusks, rice, tender sorts of vegetables, fruit juices, cottage cheese, lean milk), completely avoiding fats and animal proteins. Otherwise the treatment is symptomatic, strychnine being given when there is a fall in the arterial pressure and cardiacs in circulatory failure.

CHRONIC PANCREATITIS (PANCREATITIS CHRONICA)

Chronic pancreatitis, just as the acute form, is most commonly associated with an inflammatory process in the gallbladder, cholelithiasis, cirrhosis of the liver, cholangitis, and gastric and duodenal ulcer. A certain contributing role is played by atherosclerotic changes in the pancreatic vessels.

Pathologic anatomy. There is proliferation of connective tissue in the pancreas, enlargement of the gland and destruction of the islets of Langerhans.

Clinical picture. The disease runs a chronic course and is manifested by periodic attacks of pain resembling mild forms of acute pancreatitis. The patient complains of epigastrial and periumbilical colicky pains radiating to the left and to the small of the back, flatulence, and short breath. During a relapse the stools may be malodorous, frequent, and loose, with a large loss of fat. Microscopy of the faeces reveals a considerable number of drops of neutral fats and muscle fibres with retained striae and nuclei. The concentration of enzymes in the blood and urine is increased.

In a period of remission the patient complains of dull epigastrial pains radiating characteristically of pancreatitis. The epigastrium is tender on palpation, and a sausage-like pancreatic enlargement may sometimes be palpable.

The disease runs a prolonged course with a progressive functional inadequacy of the pancreas. The amount of pancreatic enzymes in the duodenal contents is considerably reduced, which eventually leads to fat and protein malabsorption, prolonged diarrhoea and cachexia.

In a considerable cirrhosis of the head of the pancreas obstructive jaundice may develop due to compression of the common bile duct. Palpation reveals a pear-shaped gallbladder enlargement. In affection of the islets of Langerhans glucosuria may also be encountered.

Chronic pancreatitis must be differentiated from carcinoma of the head of the pancreas, cholangitis, cholelithiasis, and enteritis. The prognosis is favourable if relapses are rare, and ominous when the latter are frequent and are attended with a prolonged emaciating diarrhoea and cachexia.

Prophylaxis and treatment. It is mandatory to prevent and treat all diseases likely to cause chronic pancreatitis, inflammatory processes in the gallbladder, cholelithiasis, cholangitis, cirrhosis of the liver, and ulcers of the stomach and duodenum. Treatment consists in dietotherapy (see "Acute Pancreatitis"), administrations of pancreatin and hydrochloric acid, and novocain blocking.

PRIMARY CARCINOMA OF THE PANCREAS (CANCER CAPITIS PANCREATIS)

Primary cancer of the pancreas takes its origin from the epithelium of the pancreatic ducts and is an adenocarcinoma in character. The tumour is usually located in the head, and less frequently in the body and tail of the pancreas. The disease mainly afflicts elderly persons.

The patient complains of a partial or total loss of appetite, debility, itching when jaundice develops, and emaciation. Pains occur if the tumour is pressing upon the solar plexus, as is the case with tumours of the body of the gland.

Inspection reveals obstructive jaundice displayed by an intense colour of the skin, with a green or black hue in particular. The development of jaundice is due to compression of the common bile duct with the tumour. As distinct from obstructive jaundice caused by occlusion of the common bile duct with a stone, there is neither antecedent rise in temperature nor biliary colic. In addition, the gallbladder, while suffering no pathologic changes, is distended by accumulated bile and is palpable as a pear-shaped elastic formation (Courvoisier's sign). The liver is enlarged due to stasis.

Obstructive jaundice gives rise to high-degree bilirubinaemia with urobilin being absent from the urine, itching and excoriation produced by scretching, infection of the skin in excoriated places, and cholaemia with symptoms of haemorrhagic diathesis (bleeding). The stools are discoloured, clayey, and contain much neutral fat. The appearance of ascites suggests compression of the portal vein by metastases to lymph nodes or by a nodular liver.

Carcinoma of the pancreas is sometimes attended with steatorrhoea, creatorrhoea, and glucosuria. Gastric secretion shows a tendency to cessation.

Jaundice in elderly persons, attended by a distended gallbladder, absence of pancreatic enzymes from the duodenal contents, and an increase in the amount of diastase in the urine should alert the physician to the possibility of a malignant newgrowth in the pancreas.

Carcinoma of the pancreas should be differentiated from a tumour of the ampulla of Vater. In the latter case jaundice is less persistent because of a periodic supply of bile to the duodenum caused by decomposition of the tumour. In carcinoma of the common bile duct the concentration of pancreatic enzymes in the duodenal contents is normal. Fibrosis of the head of the pancreas is difficult to differentiate from carcinoma of the latter since obstructive jaundice is present in both cases. Fibrosis, however, runs a prolonged course with periodic remissions whereas carcinoma progresses rapidly and causes death within 6-8 months of the initial symptoms of the disease.

Carcinoma of the body of the pancreas is less frequent than that of its head and is accompanied by intense epigastric pains, sometimes girdling in location and radiating to the back. The pains are paroxysmal and are caused by compression of the solar plexus by a tumour. Jaundice is absent, and the ascites is caused by cancerous peritonitis or compression of the portal vein by metastases to the liver, omentum, and lymph nodes.

Treatment is symptomatic. Intense pains are relieved with narcotics, and substitution therapy with insulin and lipocaine as well as a bland diet (see "Pancreatitis") are recommended in case of total resection of the pancreas.

Diseases of the Haemopoietic Organs

COMPOSITION OF THE BLOOD

The total quantity of blood in a healthy person is 5 litres, or $^{1}/_{13}$ to $^{1}/_{20}$ of the body weight. The blood consists of a liquid part known as plasma and corpuscles suspended in it. The plasma consists of water in which proteins, salts, etc., are dissolved. The blood corpuscles are erythrocytes (red blood cells), leucocytes (white blood cells), and blood platelets (thrombocytes).

Erythrocytes. Normally there are from 4,500,000 to 5,000,000 erythrocytes in 1 cu mm of blood. The erythrocytes are shaped as a disc, a bell, or a saucer averaging 7.5 μ in diameter. They supply oxygen to the tissues, thereby performing an important function in the vital activity of the body. Oxygen is conveyed to the tissues by haemoglobin contained in the erythrocytes the number of which in a healthy person is conventionally estimated at 100 or 16 g/100 ml, variations within the limits of 70-80 (12-15 g/100 ml), however, being possible. When the amount of haemoglobin is reduced but the normal number of erythrocytes is retained, the latter acquire a pale colour, and conversely, with the initial haemoglobin level being retained and the erythrocytes being reduced in number, the latter take a more intense colour.

The average life span of an erythrocyte is up to 4 months. Decomposition of dead erythrocytes normally occurs in the reticuloendothelial tissue: in the spleen, bone marrow, liver, etc.

The haemoglobin liberated in this decomposition is used for bilirubin production, while iron is used in the formation of new erythrocytes. Erythrocyte decomposition products stimulate an intensified haemopoiesis. Bone marrow supplies mature erythrocytes to the peripheral blood. When it is unable to supply mature cells to compensate for a large loss of erythrocytes the peripheral blood receives immature cells of a very pale colour due to haemoglobin deficiency (hypochromia).

In diseases of the blood one is likely to encounter erythrocytes with a diminished resistance to external influences. They acquire a peculiar shape of a spongecake, a pear, or gymnastic weights. The presence in the blood of erythrocytes of an altered form is known as poikilocytosis. The presence in the blood of erythrocytes of a variable size in the form of macrocytes and microcytes is termed anisocytosis. In certain diseases of the haemopoietic organs, nucleated erythrocytes (normoblasts) and megaloblasts (large cells having nuclei of a fine reticular structure) enter the peripheral blood from the bone marrow. The appearance of megaloblasts is evidence of transition to the embryonic type of haemopoiesis since they are found only in the blood-forming organs of an embryo. The loss of their nuclei by megaloblasts results in the formation of large nuclear-free cells—megalocytes, whose presence also indicates pathology of haemopoiesis.

Leucocytes. The number of leucocytes in 1 cu mm of blood varies from 6,000 to 8,000. They are divided into: (a) granulocytes having a granular protoplasm and (b) agranulocytes devoid of granules. Depending on their capacity to take different stains, granulocytes are subdivided into neutrophilic, eosinophilic, and basophilic. Neutrophilic leucocytes are coloured by a neutral stain.

In respect to the form of their nuclei leucocytes are subdivided into myeloblasts, promyelocytes, myelocytes, juvenile cells with a sausage-like nucleus, rodnuclear cells with an S-shaped nucleus and segmentonuclear cells with a nucleus divided into segments. Promyelocytes and myelocytes are normally absent from the peripheral blood, and their appearance indicates a severe haemopoietic disease.

The normal value of neutrophils is 60 to 65 per cent, of which juvenile cells account for 0.5 per cent and rodnuclear cells for 1-2 per cent. Eosinophils have a peculiar affinity for taking a bright-pink eosin stain (Gr. *eos*—dawn) from which they have derived their name. Eosinophils just as neutrophils exist in the form of myelocytes, juvenile, rod nuclear and segmentonuclear cells. The peripheral blood contains only segmentonuclear eosinophils which account for 2-4 per cent of all leucocytes.

The granulocytes also include basophils or mast cells. They are called basophilic because of their affinity for basic dyes which lend them a violet colour with a lilac hue.

The following agranulocytes are distinguished: (a) monocytes, large cells with a large nucleus, a fine spotted structure, and a protoplasm taking a bluish stain; their value is normally equal to 3-6 per cent; (b) lymphocytes having a round or oval nucleus rich in chromatin; they stain violet and are surrounded with a thin blue rim of protoplasm.

Lymphocytes usually account for 25-30 per cent of the total number of leucocytes. In certain diseases the peripheral blood contains juvenile lymphocytes known as lymphoblasts which are normally found only in lymph nodes.

The blood contains 250,000 to 400,000 thrombocytes in 1 cu mm. They play a highly important role in coagulation of the blood. When allowed to stand in a vessel the plasma forms the upper layer, while the lower layer is formed of blood corpuscles: leucocytes, erythrocytes and thrombocytes. Normally the ratio of plasma to formed elements is 55 : 45. If no substances inhibiting coagulation are added to effused blood the latter shortly turns into a blood clot consisting of fibrin with captured formed elements. When allowed to stand for a certain time this clot produces serum.

In an adult haemopoiesis takes place in the bone marrow, lymph nodes, and spleen. The bone marrow produces granulocytes, erythrocytes, and thrombocytes. Lymphocytes are formed of lymphoblasts in the lymph nodes and follicles of the spleen. Monocytes are formed of reticuloendothelial cells in the spleen, bone marrow and lymph nodes. In adults these three systems exist independently and mature cells do not transform into each other. The stem cell from which all blood corpuscles develop is known as the haemocytoblast. The theory of the development of all blood corpuscles from a common stem cell has been proposed by the Russian scientists N. Kuskov, V. Obraztsov, A. Maximov and A. Kryukov and is known as the unitarian theory. In an adult person, active marrow fills the epithyses of all long bones and flat bones—the sternum, skull, ribs and spinal column. In the diaphyses the yellow bone marrow is inactive, but in leukaemia and severe anaemia it becomes active.

In the embryo, haemopoiesis in the marrow begins from the 4th month of life. Before this time it goes through the hepatic and then splenic stages. In diseases of the haemopoietic system an intensified blood formation begins above all in the bone marrow, and later foci of haemopoiesis appear in the liver, spleen, and lymph nodes. The active bone marrow contains leucoblasts and erythroblasts. Leucoblasts constitute four-fifths of the bone marrow and produce polymorphonuclear granulocytes from myeloblasts through the stages of promyelocytes, myelocytes, and juvenile cells. The granulocytes are very quickly destroyed in the process of phagocytosis. The erythroblasts constitute one-fifth of the active bone marrow and give origin to erythrocytes through the stages of proerythroblasts, erythroblasts, normoblasts and reticulocytes. The peripheral blood contains only mature erythrocytes but even under normal conditions one may encounter reticulocytes, i.e., cells which have lost their nucleus but retained the reticular structure of the protoplasm. They number 5 to 10 per 1,000 erythrocytes.

Upon complete cessation of the activity of bone marrow, mature erythrocytes disappear from the peripheral blood within 2 months. The megaloblastic type of haemopoiesis is retained in the foetus up to 4 months.

Normal maturation of erythrocytes is connected with the supply of an antianaemic substance whose deficit leads to the formation of megaloblasts and megalocytes. Normal haemopoiesis also depends on the supply of iron which is necessary for haemoglobin production. In case of an iron deficit erythrocytes of a normal structure, but of a pale colour, are formed. Blood platelets develop from megakaryocytes; they have no nuclei, are not stable in the peripheral blood, readily stick together, and play an important role in coagulation of the blood.

Regulation of haemopoiesis. Normal haemopoiesis is made possible by the supply of an antianaemic substance (vitamin B_{12}), protein, iron, and erythrocyte decomposition products. Highly important for the regulation of haemopoiesis are hormones of the thyroid gland, the anterior lobe of the pituitary body, genital glands, and adrenals, as well as salts of copper, arsenic, and ascorbic acid. The nervous system plays an important role in the regulation of haemopoiesis. The bone marrow is provided with a rich network of nerve branches (interoceptors) whose stimulation is transmitted to the central nervous system. S. Botkin wrote in his time that the central nervous system has a special centre governing haemopoiesis and disintegration of blood corpuscles. The studies carried out by K. Bykov and his associates as well as those by V. Chernigovsky have proved the all-important role of the nervous system in haemopoiesis. M. Konchalovsky, G. Lang and N. Strazhesko, in contradiction to Virchow's organolocalistic theory in haematology, developed a theory of neurohumoral regulation of haemopoiesis, thereby substantiating functional haematology and supplementing S. Botkin's theory of the regulating role of the central nervous system in haemopoiesis.

ANAEMIA

Anaemia (Gr. *an*—negative particle, *haima*—blood) literally means "bloodlessness" but actually there is only a diminution in the number of erythrocytes and in the haemoglobin level per unit volume. It is necessary to distinguish between genuine anemia and a reduction in the number of erythrocytes and amount of haemoglobin as a result of dilution of the blood in the period of abatement of oedema when tissue fluid enters the blood stream (hydraemia). For this purpose, one has to determine the ratio of erythrocytes to blood plasma which is normally equal to 45 : 55, while in hydraemia it is altered even more in favour of the plasma. In addition, it should be borne in mind that genuine anaemia may be overlooked if the patient suffers from profuse diarrhoea and persistent and copious vomiting which cause haemoconcentration.

Anaemia causes oxygen deficiency in tissues (hypoxia) which is manifested by dyspnoea, palpitation, distressing sensations in the heart region. Oxygen deficiency, however, depends not only on the degree of anaemia but also on the rapidity of its progression. With anaemia progressing slowly the body becomes adapted to altered conditions of existence, because underoxidized metabolic products enter the blood stream and, acting on the central nervous system, produce tachycardia, acceleration of the blood flow, an increase in the minute volume of the heart and in the mass of circulating blood at the expense of its reserves in the blood depots.

Classification of Anaemia

The modern classification of anaemic states is based on M. Konchalovsky's proposal to divide this condition into three groups: (a) anaemia due to acute and chronic blood loss; (b) anaemia caused by disturbances in haemopoiesis; (c) anaemia resulting from intensified destruction of red blood cells (haemolysis).

ANAEMIA DUE TO BLOOD LOSS OR POSTHAEMORRHAGIC ANAEMIA (ANAEMIA POSTHAEMORRHAGICA)

Acute Posthaemorrhagic Anaemia

This anaemia results from a blood loss due to external traumas (wounds), haemorrhages from the gastrointestinal tract (gastric and duodenal ulcer, cancer and polyposis of the stomach and intestine, haemorrhoids, cirrhosis of the liver, etc.) and from the lungs, rupture of a fallopian tube in extrauterine pregnancy, etc.

Clinical picture. The disease sets in with a sudden pallor, the skin being moist from cold and sticky sweat. The patient sometimes faints or complains of dizziness, tinnitus, palpitation, intense thirst, and lassitude. Less frequently he becomes excited. The pulse is frequent and small and there is hypotension. In case of a considerable blood loss a systolic murmur is audible over the apex of the heart and over the pulmonary artery, and the respiratory rate is quickened.

Blood picture. A blood loss is immediately followed by a reduction in the number of erythrocytes and in the level of haemoglobin. Later this reduction progresses considerably due to absorption of tissue fluid into the blood stream and dilution of the blood (hydraemia). In 4-5 days a large number of juvenile erythrocytes (reticulocytes) are delivered into the peripheral blood from the bone marrow (up to 10-12 per cent instead of the normal 0.5-1.0 per cent). The red blood shows normochromic (normal colour index) microcytic anaemia with poikilocytosis and anisocytosis. The number of leucocytes is increased, juvenile cells and even myelocytes making their appearance. The iron content of the blood serum is reduced. With a considerable loss of blood the bone marrow in flat bones shows a fairly high activity, while the yellow marrow of the tubular bones turns red, i.e., becomes capable of haemopoiesis. The normal blood values are completely restored within 2-3 weeks. This term may be shortened or lengthened depending on the amount of blood lost, the condition of adaptational mechanisms, standards of care ot the patient, the diet and efficacy of treatment. In massive haemorrhage, sudden death occurs due to shock or collapse.

Chronic Posthaemorrhagic Anaemia

Frequent and even insignificant blood losses occurring in the same diseases as an acute blood loss and concurrent with an inadequate function of the bone marrow, as well as a single but massive haemorrhage, may lead to chronic anaemia when the iron reserves in the body become exhausted.

Clinical picture. The patient's complaints vary in character with the degree of anaemia and are manifested by general debility, frequent dizziness, fatigability, and dyspnoea. There is pallor, the skin colour has a wax-like hue, the face is puffy, the lips bloodless, the shanks pasty or oedematous, emaciation being absent if the blood loss has not been caused by a tumour. Sometimes an increased deposition of fat in subcutaneous cellular tissue is noted. Examination of the cardiovascular system reveals a cardiac systolic murmur and a humming-top murmur over the veins of the neck.

Blood picture. In the peripheral blood the number of erythrocytes and the level of haemoglobin are reduced, the colour index is lowered to 0.5-0.6, with poikilocytosis, anisocytosis, and microcytosis, the white blood showing leucopenia, relative lymphocytosis, moderate thrombocytopenia. The number of reticulocytes is increased (2-4 per cent). In the bone marrow there is hyperplasia of erythroblastic tissue, the total number of erythroblasts is increased, their maturation, however, being slowed-down, as a result of which some of them are destroyed or enter the blood stream in the form of microcytes.

Treatment. In acute blood loss the cause of bleeding, if it is known, should be immediately eliminated, and the bleeding arrested. The patient is placed on his back with his feet raised slightly in order to ensure normal blood supply to the brain, and is given much liquid to drink. For internal haemorrhages haemostatic agents are prescribed: 10 ml of a 10 per cent calcium chloride solution, 15 ml of a 10 per cent sodium chloride solution, or 30-50 ml of blood plasma (or dry plasma dissolved in physiologic solution) intravenously, and 10 ml of horse (antitetanic, antidiphtheric) serum intramuscularly. In such cases intravenous infusions of fresh and banked donor blood as a haemostatic (100-120 ml) and substitution (400-500 ml) factor are also helpful. Vitamin K (vicasol) is also prescribed.

In chronic blood loss, whenever possible, treatment of the principal disease responsible for bleeding is carried out by means of surgical interference (resection of the stomach, extirpation of a fibromatous uterus, excision of haemorrhoid piles). Erythropoiesis is stimulated by repeated haemotransfusions in doses of 200-300 ml at intervals of 5-7 days. Other remedies are large doses of hydrogen-reduced iron (Ferrum hydrogenium reductum) in a dose of 1 g 3-4 times daily, Blaud's pills with ascorbic acid after a meal. In gastric achylia preparations of iron are given together with diluted hydrochloric acid since the latter contributes to iron absorption. The acid is given in a dose of 20-25 drops per os. Treatment with iron preparations is continued for 1-2 months. Ferkoven (a preparation containing sucrose, iron and cobalt salt of gluconic acid) in a dose of 5 ml daily or every other day is administered parenterally. It is introduced slowly in 15 ml of a 40 per cent glucose solution, the course of treatment consisting of 10-15 infusions. Ferkoven quickly raises the haemoglobin level in posthaemorrhagic anaemia. The diet must be rich in proteins and iron (up to 120 g of proteins daily) and fat intake should not exceed 40 g in 24 hours. Fresh greens, fruit, vegetables as well as foodstuffs containing vitamins A_1, B_1, B_{12} and C are also recommended.

ANAEMIA DUE TO DISTURBANCES OF HAEMOPOIESIS

Iron Deficiency Anaemia

Iron deficiency anaemia results from a deficiency of iron in the body due to an acute or chronic blood loss and increased consumption or poor absorption of iron. In this condition, the iron content of the blood serum is reduced. Anaemia may be hypochromic, with a low colour index.

Chlorosis or Geern Sickness

Chlorosis is a rare disease and is encountered only in young girls in the period of puberty.

Aetiology and pathogenesis. The aetiology of this disease has been inadequately studied so far. S. Botkin and G. Zakharyin regarded chlorosis as neuroendocrine disorder. A certain importance was also attached to congenital factors such as congenital iron deficiency in the body, as well as to environmental factors—the diet, a history of infectious diseases, poor hygienic conditions of life (insufficient fresh air, lack of physical exercises, etc.). Chlorosis was particularly frequent in the past in private educational establishments. Over the past few decades the incidence of chlorosis has sharply declined because of the growing interest of girls in physical culture and sports. Apparently an important cause of chlorosis is a disturbance in iron absorption in the body, which is provoked by the onset of the menses. Simultaneously there is underdevelopment and impairment of ovarian and uterine function which results in menstrual disturbances.

Clinical picture. The patient complains of sleepiness, lassitude, tinnitus, headaches, dizziness, predisposition to fainting, irritability, palpitation, breathlessness, constipation and heartburn. The appetite is usually decreased and perverted (pica chlorotica), the patient showing a craving for drinking vinegar, eating coal and chalk. The menses are scant, less frequently profuse and irregular. The colour of the face is greenish-yellow (the term chlorosis is derived from the Greek *chloros*—green), the visible mucous membranes are pale, the skin has an alabaster colour and is oedematous or pasty, especially on the face and shanks. The pulse is usually rather frequent, the arterial pressure is low, and the heart borders are within normal limits. A systolic murmur is audible over all orifices of the heart and particularly over the pulmonary artery, while a humming-top murmur is heard over the jugular vein on the neck, caused by a reduced viscosity of the blood and an accelerated blood flow. In the peripheral blood there is a reduction in the haemoglobin level and, to a lesser extent, in the number of erythrocytes, i.e., hypochromic anaemia (colour index being within limits of 0.5-0.6), with anisocytosis and poikilocytosis. The number of reticulocytes is increased but slightly. The iron content of the blood serum is reduced, hypoproteinaemia (mainly at the expense of albumins) and hypocholesteraemia are noted. The stomach secretion is increased, the menses are scant and are attended with severe pains.

Late Chlorosis (chlorosis tarda)

This form of chlorosis was for the first time described by M. Konchalovsky. It is encountered in elderly women before the onset of the menopause. It is also occurs at ages of 30-45 years, usually after childbirth, menstruations, haemorrhoid bleeding, in a steep reduction of gastric secretion, when iron absorption is considerably impaired. Late chlorosis is also encountered in pregnancy in which the maternal body is supplying the foetus with its own reserve iron, without adequate replenishment of this loss.

Clinical picture. This is almost identical with that of juvenile chlorosis. The patient complains of fatigability, dizziness, tinnitus, chilling of the extremities, chillness and a sensation of creeps in the skin. The skin acquires an alabaster colour, aphthae appear on the tongue, the tongue papillas become atrophied and histamine-resistant achylia is noted in the stomach. The patient has a perverted appetite, with a craving for chalk, earth, dental powder (pica chlorotica). There is atrophic disturbance, the hair becomes brittle and comes out in tufts, the nails are extremely fragile and deformed, sometimes concave (spoon-shaped nails—koilonychia). The level of haemoglobin and erythrocytes is dimnished, the latter's colour being very faint and the leucocytes being unchanged.

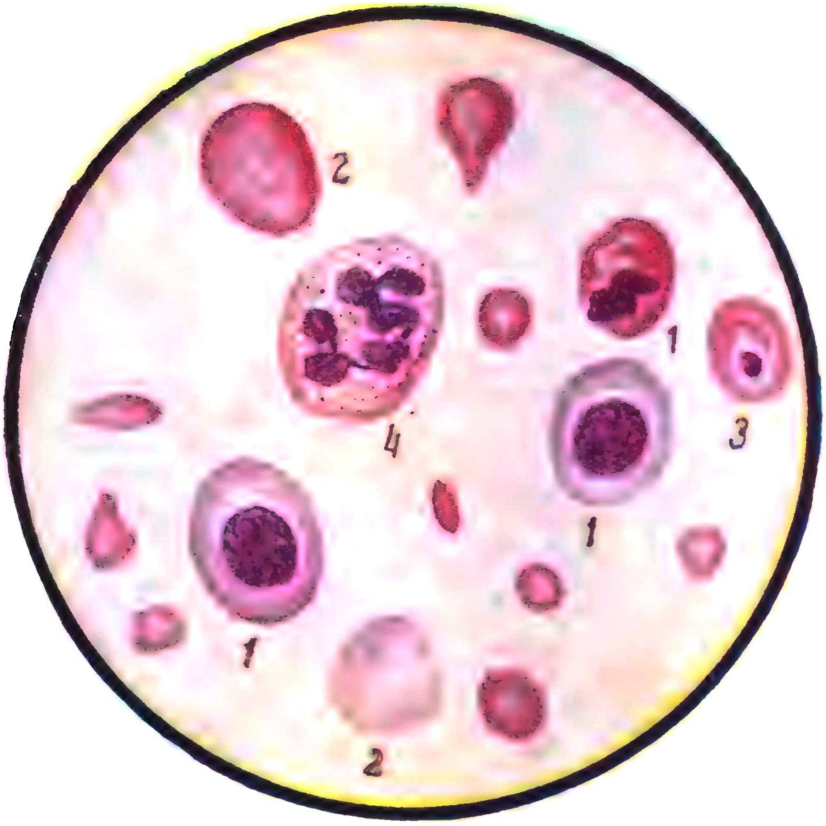


Fig. 70. Blood picture in pernicious anaemia
1—megaloblasts; 2—megalocytes; 3—megalocytes with Jolly body; 4—segmentonuclear erythrocytes

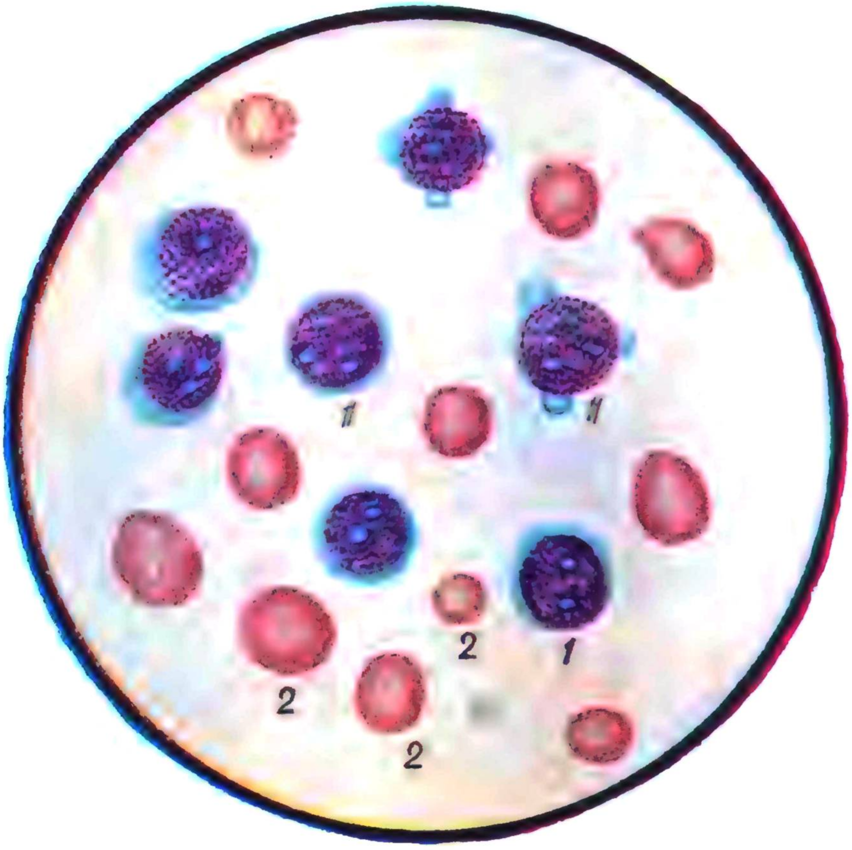


Fig. 71. Blood picture in acute myelosis
1—myeloblasts; 2—erythrocytes

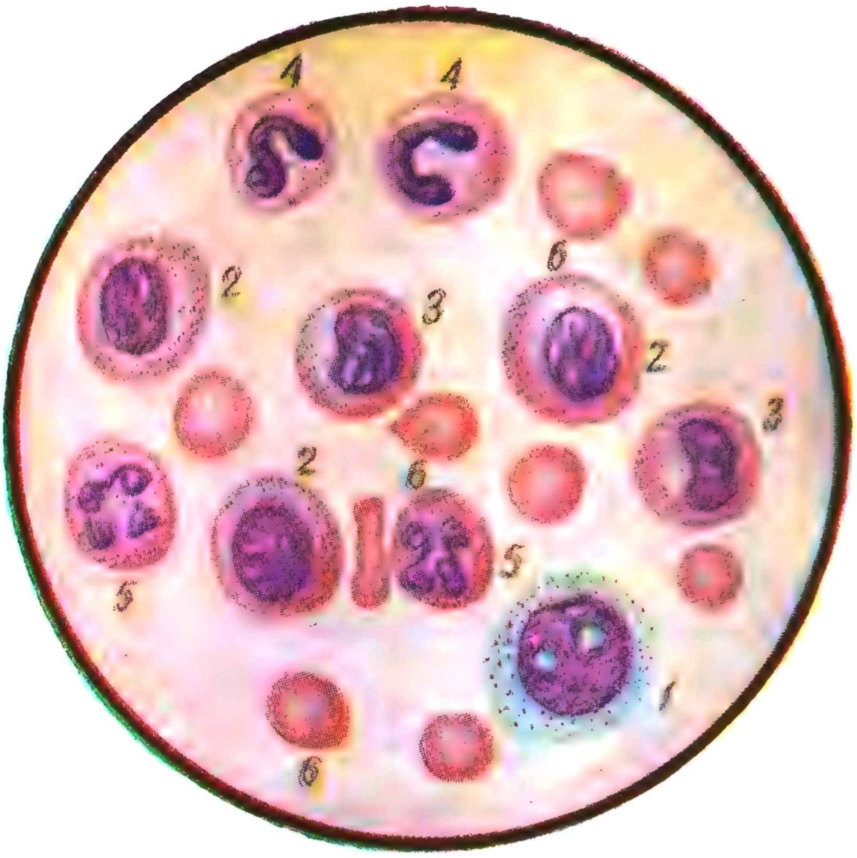


Fig. 72. Blood picture in chronic myelosis

1—promyelocyte; 2—myelocytes; 3—juvenile neutrophils; 4—rod neutrophils; 5—segmentonuclear neutrophils; 6—erythrocytes

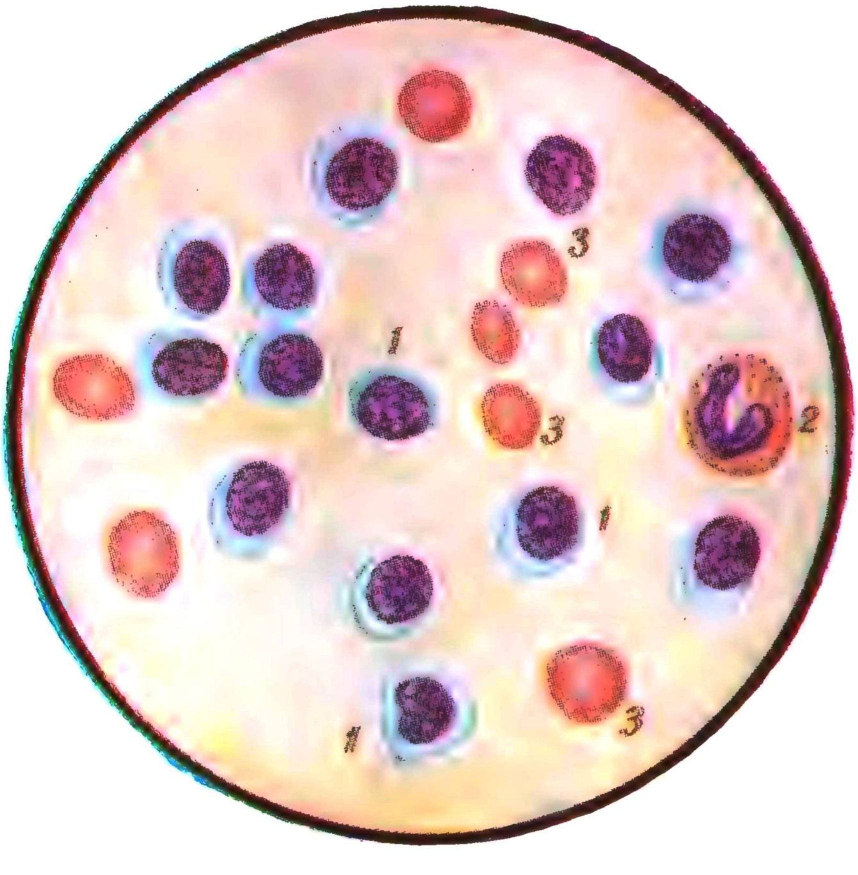


Fig. 73. Blood picture in chronic lymphadenosis

1—lymphocytes; 2—rod neutrophil; 3—erythrocytes

Treatment of chlorosis. Highly important are hygienic habits, supply of fresh air, a nutritious diet rich in iron (meat, liver, vegetables, etc.). Iron is prescribed per os in a dose of 1 g to be taken 3-4 times daily in combination with dilute hydrochloric acid. Iron treatment should be continued for a long time. Also very helpful is haemostimulin (a preparation containing cattle blood, iron lactate, copper sulphate and glucose.—*Ed.*) administered in a dose of 0.6 g 2-3 times daily. With a considerable anaemia transfusions of blood (200-300 ml) and erythrocyte mass (200 ml) are indicated. In chlorosis subcutaneous injections of folliculin or synestrol are recommended in order to stimulate ovarian function.

Achlorhydric Iron Deficiency Anaemia (Chloranaemia, Achylic Anaemia)

Achylic anaemia is mainly encountered in women, less often in men. The leading pathogenetic factor is gastric achylia, since iron may be absorbed in the intestine only in the form of ferrochloride, and the latter's formation is possible only if hydrochloric acid is present in the gastric juice. Another factor contributing to gastrogenic anaemia is insufficiency of iron in food, especially in case of its increased excretion (profuse menstruations, haemorrhoids, etc.) and utilization, as is the case in pregnancy, lactation, etc. Gastrogenic anaemia is encountered after gastroctomy and in certain diseases (chronic enterocolitis, etc.) when iron absorption is impaired and the iron level in the blood serum is considerably reduced.

Clinical picture. The blood picture of gastrogenic anaemia largely resembles that of chlorosis, the only difference being its macrocytic character, with poikilocytosis, anisocytosis, and hypochromia being less marked than in chlorosis. There is also leucopenia with a relative lymphocytosis.

Gastrogenic anaemia must be differentiated from anaemia seen in cancer, late chlorosis, posthaemorrhagic and pernicious anaemia.

The prognosis is favourable and the capacity for work is impaired but slightly.

Treatment is the same as that for all other iron-deficiency anaemias (see "Treatment of Chlorosis").

B_{12} (FOLIC) DEFICIENCY ANAEMIA

Pernicious Anaemia (anaemia perniciosa)

Aetiology and pathogenesis. Pernicious anaemia was for the first time described by Addison (1855) and later was dealt with in greater detail by Biermer (1868-1872); therefore, it has been named Addison-Biermer's disease. This type of anaemia is called pernicious because it is known to have run an extremely severe course prior to the introduction of raw liver therapy.

Various theories have been advanced to explain the causes of pernicious anaemia. At first its cause was attributed to gastric achylia and a dietary deficiency. However, after a brilliant therapeutic effect had been obtained by raw liver therapy (Minot and Murphy, 1925) it was established that raw liver is capable of normalizing the haemopoietic function of the bone marrow. Later Castle (1929) succeeded in restoring the normal function of the bone marrow in pernicious anaemia by feeding the patient daily with raw beef preliminarily treated with gastric juice of a healthy person. This prompted him to assert that an intrinsic factor is produced in the stomach, which by interaction with an antianaemic extrinsic factor derived from raw meat forms an antianaemic haemopoietic factor contributing to the maturation of juvenile erythrocytes. It was elucidated later that the extrinsic factor is vitamin B_{12} (cyanocobalamine), while the intrinsic factor is gastromucoprotein produced by additional cells in the fundal part of the stomach. Vitamin B_{12} binds with gastromucoprotein to form an unstable combination which, while passing through the intestinal mucosa, is liberated from gastromucoprotein and, binding with α_1-globulin, is deposited in the liver, from where it is supplied into the blood stream whenever required, stimulating the activity of the bone marrow. In recent time it has been supposed that vitamin B_{12} is acting on the bone marrow through folic acid which is converted by it into folinic acid.

It is assumed that pernicious anaemia develops as follows: in the absence of gastromucoprotein, vitamin B_{12} fails to be derived from food, no antianaemic substance is produced, with a resulting impairment of haemopoiesis. In the bone marrow, therefore, the maturation of erythroblasts is retarded, while the formation of megaloblasts and megalocytes occurs, which is characteristic of embryonic haemopoiesis. Consequently, pernicious anaemia is due to vitamin B_{12} deficiency resulting from cessation of gastromucoprotein production. At the same time there are cases of pernicious anaemia with a normal gastromucoprotein secretion but inadequate vitamin B_{12} supply with food. The cause of disturbance in gastromucoprotein production is uncertain and cannot be traced to gastric achylia, cancer of the stomach or gastritis. Most commonly it is associated with atrophy of the gastric mucosa and polyposis of the stomach.

Pathologic anatomy. All organs are anaemic; there is fatty degeneration of the heart, liver, kidneys, atrophy of the gastrointestinal mucosa, and degenerative changes in the posterior and lateral columns of the spinal cord. There are haemosiderin deposits in the bone marrow, liver, lymph nodes, kidneys, and spleen. The diaphyses of the tubular bones are activated and acquire a crimson colour. The bone marrow contains a large number of megaloblasts. There are foci of extramedullary haemorrhage in the liver, spleen and kidneys. The iron liberated in decomposition of erythrocytes is deposited in Kupffer's cells of the liver, and in the spleen, and lungs which become rusty in colour.

Clinical picture. Pernicious anaemia is insidious in onset and sometimes manifests itself suddenly after influenza, tonsillitis, a physical or mental trauma.

The disease is the most frequent in persons older than 50. The characteristic complaints are debility, dyspnoea, palpitation, precordial pains, a sensation of bright specks appearing before the eyes, and a burning of the tongue. A burning sensation in the oesophagus is also encountered as a result of its being inflamed, particularly upon ingestion of sour food. Vomiting and diarrhoea may likewise occur. There is a tingling sensation in the skin, numbness of the extremities, a disorder in the gait, and girdle pains in the lumbar region. The patient's appearance is highly characteristic: the skin is of a lemon-yellow colour, the mucous membranes are pale, the scleras are subicteric, and the face is oedematous. Far from being emaciated, the patient sometimes has an increased deposition of fat in the subcutaneous cellular tissue. The tongue is clean, bright-red in colour, smooth, with obliterated papillas (Hunter's glossitis). Sometimes it is covered with aphthous vesicles or small superficial ulcers. The gastrointestinal tract exhibits histamine-resistant gastric achylia, sometimes diarrhoea with malodorous stools containing much stercobilin. The liver is enlarged and soft on palpation. There is an insignificant enlargement of the spleen. The urobilin reaction in the urine is positive, and the bilirubin content of the blood is elevated. The cardiovascular symptoms are dyspnoea, precordial pains, tachycardia, hypotension, a systolic murmur of anaemic and partly muscular character, because the heart muscle is undergoing dystrophy (tiger heart), a humming-top murmur over the bulb of the jugular vein. A decreased viscosity of the blood and a faster blood flow are also noted. Changes in the nervous system are manifested by sclerosis of the posterior and lateral columns of the spinal cord consequent upon demyelinization and degeneration of the cylinders of the central neurons. This affection is known as funicular myelosis and is manifested by a combination of spastic and spinal paralysis and tabetic symptoms with pathologic reflexes (knee jerk reflex, Achilles tendon reflex, Babinski's and Rossolimo's reflexes), clonuses, girdle pains accompanied by a disturbance of vibratory and deep sensibility and retention of superficial sensation, and involuntary urination and defecation. The body temperature is elevated, sometimes to 39-40°C, which is accounted for by an intensified decomposition of the blood cells and rejuvenation of the bone marrow. In certain cases there is deafness which is due to neuritis of the acoustic nerve and blindness associated with atrophy of the optic nerve.

The disease runs its course with periodic remissions and recidivations. In periods of remission the number of reticulocytes is increased and during recidivations, reduced. The white blood exhibits leucopenia, an increase in leucocytes and a diminution in blood platelets. Before the advent of liver therapy the disease was truly malignant in character and usually ended fatally within 2-3 years of its being identified. After the introduction of liver therapy suggested by Minot and Murphy, the course of pernicious anaemia has changed considerably. A patient given regular liver therapy retains his capacity for work for decades.

Blood picture. The greatest changes are seen in the blood (Fig. 70). The colour index is above or close to 1 and even in periods of remission is not lower than 0.8-0.7. The erythrocytes are supersaturated with haemoglobin. The blood is found to contain erythroblasts (nucleated erythrocytes), macrocytes, megaloblasts, megalocytes—nuclear-free giant erythrocytes. Haemopoiesis is of the embryonic type. In exacerbation of the disease the number of reticulocytes in the peripheral blood is sharply reduced while their increase forebodes an oncoming remission. The peripheral blood may show poikilocytosis, anisocytosis, erythrocytes with Jolly bodies and Cabot rings. The number of granulocytes is diminished. The blood may be found to contain giant multinuclear neutrophils and sometimes even juvenile cells and myelocytes. Thrombocytes are reduced in number, but haemorrhagic symptoms are, as a rule, absent. Examination of the bone marrow aids diagnosis. It shows hyperplasia of erythropoietic tissue, an increase in the number of erythroblasts and megaloblasts, a retardation of the maturation of erythroblasts due to vitamin B_{12} deficiency and a diminution in the amount of folic acid.

Treatment and prophylaxis. The patient must be ensured hygienic conditions of life and safeguarded against physical and nervous overstrain. In periods of relapses he is given 200 g of minced lean calf liver daily. To make it more digestible, liver is served with onions, pepper and garlic or is slightly roasted. The liver is very rich in iron and vitamins, mainly vitamin B_{12}. Also very helpful are liver extracts among which the most famous is the Soviet preparation campolon obtained from cattle liver. The latter is administered intramuscularly in doses of 2 ml every other day, sometimes in a massive dose of 16-20 ml. Within 5-6 days of campolon injection there is an increase in the number of reticulocytes to 20-30 per cent (reticulocytic crisis) followed by a growth in the level of haemoglobin and number of erythrocytes and by normalization of the red blood composition within 4-6 weeks. Folic acid is also prescribed per os or parenterally in a dose of 30-60 mg daily. Another remedy is antianaemin containing cobalt which also has an antianaemic effect (in doses of 2-4 ml daily until a haematologic remission).

The most efficacious is vitamin B_{12} which is injected intramuscularly daily or every other day in doses of 100-200 γ. Of late vitamin B_{12}, biopar, in tablet form in combination with gastromucoprotein, as well as bifarcan and cytobion *M* have been used. After vitamin B_{12} has taken effect and the anaemia has become hypochromic, iron is prescribed in doses of 3-4 g daily to be taken with hydrochloric acid. It is highly important to resort to prophylactic vitamin B_{12} treatment (in a dose of 30 γ once weekly) to prevent a relapse. For prophylactic purposes observation should be instituted over persons who have sustained gastric resection or suffer from organic gastric achylia so as to notice changes in the red blood in due time. It is particularly important to maintain such patients on a diet containing nutritious proteins—meat, eggs, fruit, vegetable juices—and give them prophylactic antianaemic therapy (200 g of raw liver once weekly).

When untreated, the patient falls into a comatose state, the haemoglobin level is considerably reduced, the erythrocytes are extremely low in number, other salient features being oedema, jaundice and somnolence. The patient may be recovered from a comatose state by repeated blood transfusions, using the drip method in particular, and by intramuscular injections of large doses of liver preparations.

Pernicious-Like Anaemia Due to Helminthic Diseases

Pernicious-like anaemia sometimes occurs in broad tapeworm infection (bothriocephalus anaemia). S. Botkin believed that in such cases anaemia is due to reflex stimulations of the brain conveyed from the gastrointestinal tract with its multiple nerve endings. Some authors assert that helminthic invasion causes a disturbance in vitamin B_{12} absorption. Others note that the parasite needs vitamin B_{12} for its nourishment. It is most probable that bothriocephalus anaemia is caused by destruction of vitamin B_{12} in the intestine by the parasites. This is evidenced by the fact that expulsion of the broad tapeworm is followed by complete disappearance of anaemia. In other cases, in spite of elimination of the parasites the anaemia persists and tends to progress. Apparently the broad tapeworm may provoke genuine pernicious anaemia. The pernicious-like form of anaemia runs a milder course, and is not acompanied by atrophic changes in the stomach and intestine, the gastric secretion being normal or deranged only slightly. The nervous system suffers no serious damage either.

Pernicious-Like Anaemia of Pregnancy

Pernicious-like anaemia usually develops in the 4th-5th month of pregnancy as a result of draining the maternal antianaemic principles—vitamin B_{12} and folic acid, which are utilized in foetal haemopoiesis.

ACHRESTIC ANAEMIA

Achrestic anaemia, i.e., anaemia due to inability to utilize the antianaemic principle resembles pernicious anaemia in many respects. Hyperchromic megalocytic anaemia is also present. Its aetiology is unknown. Achrestic anaemia differs from pernicious anaemia in causing no damage to the gastrointestinal tract, there is no achylia, nor Hunter's glossitis. The blood picture in periods of relapse, however, is identical with that of pernicious anaemia. In the bone marrow haemopoiesis megaloblasts at different stages of maturation predominate. The disease tends to progress and the prognosis is usually ominous, the patient dying within two years.

Antianaemic therapy usually employed for pernicious anaemia is of little effect in such cases. Regular transfusions of blood are indicated.

APLASTIC ANAEMIA OR PANMYELOPHTHISIS (ANAEMIA APLASTICA, PANMYELOPHTHISIS)

Aplastic anaemia, otherwise known as panmyelophthisis (wasting of the bone marrow) is a haemopoietic disease producing complete aplasia of the bone marrow.

Aetiology and pathogenesis. It is not always possible to identify the cause of aplastic anaemia. It is most commonly encountered in intoxications with benzene, petrol, arsenic, lead, etc., and is also consequent on exposure to radioactive substances, x-rays, radium, the effects of certain medicines (myelosan, sulphonamides, salvarsan, etc.). Of late evidence has been gained in favour of aplastic anaemia being due to accumulation in the body of antierythrocytic, antileucocytic and antithrombocytic substances causing anaemia, leucopenia and thrombocytopenia.

As a rule, complete wasting of the bone marrow occurs relatively rarely. It is only a diminution of all nuclear elements that is usually seen. This is known as hypoplastic anaemia which, as distinct from aplastic anaemia, runs a longer course, but finally progresses to aplasia. In this respect hypoplastic anaemia may be considered to be the initial phase of aplastic anaemia.

Pathologic anatomy. There are fat deposits in the subcutaneous cellular tissue, omentum and epicardium, which are due to a reduction in the oxidative processes occurring in the body. The flat bones, vertebrae, and long bones contain no bone marrow, the latter being supplanted with fat. The heart, liver and kidneys undergo fatty dystrophy. Haemorrhages of variable size are notable.

Clinical picture. There is general debility, fatigability, dizziness, dyspnoea, palpitation, suggilations, haemorrhages from various organs. The skin and mucous membranes are pale and the face is puffy. Tachycardia and hypotension are encountered. A systolic murmur is audible over the heart apex. The liver, spleen, and lymph nodes are not enlarged, and the body temperature is often subfebrile.

The cardinal symptoms of aplastic anaemia are haemorrhages, necrosis and ulceration. Anaemia develops rapidly and runs a violent course progressing to a high degree. No juvenile erythrocytes occur in the peripheral blood and in a sternal needle biopsy because wasting of the bone marrow produces its paralysis with complete disappearance of all cellular elements.

Predisposition to bleeding is the second most serious sign of aplastic anaemia. There are petechiae and ecchymoses in the skin and mucous membranes. Nasal and gastrointestinal haemorrhages, haematuria and thrombocytopenia occur. Coagulation of the blood is unimpaired. Necrotic ulceration is mainly localized to the tonsils, palate and mucous membrane of the cheeks. The gums are succulent and bleeding. Similar ulcerations are encountered on the skin and the intestinal follicles. Simultaneously with a considerable reduction in the haemoglobin level and in the number of granulocytes, i.e., neutrophils and eosinophils, there is a steep decline in phagocytic function, and death due to sepsis ensues. The lymphocytes account for 80-90 per cent of all leucocytes. In the terminal stages an exhaustive fever sets in.

Prognosis is unfavourable. The patient dies within 3-6 months. The outlook is relatively brighter in hypoplastic anaemia, particularly when its aetiology is known (benzene, x-rays, isotopes, etc.), since elimination of toxic external factors may arrest the disease process.

Prophylaxis. The first step is strict observance of safety rules on jobs involving exposure to x-rays, radium and radioisotopes, vigorous protective measures in industries with occupational health hazards (benzene, arsenic, lead, etc.). Observance of safety rules, periodic medical examination combined with regular blood tests in persons exposed to occupational health hazards are reliable measures of prophylaxis of aplastic and hypoplastic anaemia.

Treatment. Systematic transfusions of blood in doses of 250-300 ml, erythrocyte mass in a dose of 125-150 ml once in every 3-4 days, leucocyte and thrombocyte mass are recommended. Of late, transplantation of bone marrow has been used. Usually from 3 to 6 injections of 20 to 150 ml of sternal or iliac punctate obtained from healthy persons are made into the patient's sternum. Also indicated is treatment with ACTH (20 U twice daily), prednisone, prednisolone in doses of 10 mg 2-3 times daily. Other remedies are iron, vitamins of B-complex for a long period, folic acid in doses of 20-25 mg three times daily. Vitamins C and K (formulas Nos. 159 and 159*a*) are also administered for bleeding in addition to transfusions of blood plasma, whole blood or erythrocyte mass.

ANAEMIA DUE TO INCREASED DESTRUCTION OF ERYTHROCYTES (HAEMOLYTIC ANAEMIA)

Anaemia resulting from an intensified destruction of erythrocytes, otherwise known as haemolytic anaemia, is divided into two types: (1) that mainly caused by intracellular haemolysis of erythrocytes, and (2) that caused by intravascular haemolysis.

Congenital Haemolytic Anaemia (anaemia haemolytica congenita)

This form of anaemia may be congenital and is hereditary.

Aetiology and pathogenesis. Various views have been advanced to explain the causes of intensified haemolysis. Some believe that haemolysis is caused by an increased haemolytic function of the spleen, others attribute it to the inadequacy of erythrocytes readily disintegrating in the spleen, still others, to a congenital absence from the body of antihaemolysins which normally neutralize spleen haemolysin (lysolecithin).

The disease is essentially due to a reduction in the resistance of erythrocytes resulting in their increased haemolysis in organs rich in reticuloendothelial cells: in the spleen and partly the bone marrow, the liver and lymph nodes. A reduction in erythrocyte resistance is established with the aid of a physiologic solution of various concentration. Normal erythrocytes do not undergo haemolysis in a physiologic solution (0.85 per cent) as well as in a 0.5-0.3 per cent sodium chloride solution. With a reduction in the resistance of erythrocytes, however, haemolysis begins from the concentration of 0.6-0.4 per cent. At first the erythrocytes influenced by hypotonic common salt solutions become swollen, grow in size and acquire a spherical shape (spherocytes). Later the distended coat of the spherocytes ruptures and haemolysis ensues. Haemolysis is accompanied by jaundice, an increase in the bilirubin content of the blood, and in urobilin of the urine. Because of their decreased resistance the life span of erythrocytes does not exceed 7-8 days (instead of the normal 120-130 days). This entails increased bone marrow haemopoiesis and intense drainage of immature erythrocytes—normoblasts and particularly reticulocytes—from the bone marrow into the peripheral blood.

Pathologic anatomy. Haemosiderin, a product of erythrocyte decomposition is deposited in the spleen, bone marrow, lymph nodes, and Kupffer's cells of the liver. The bone marrow is red and hyperplastic. The gallbladder sometimes contains bilirubin stones.

Clinical picture. The first symptoms of the disease appear in childhood, less frequently in adulthood. Initially only an insignificant icterus of the scleras and mucous membranes is notable. Later nasal, gastrointestinal haemorrhages occur, sometimes attacks of hepatic colic caused by bilirubin stones.

The disease runs its course with intermittent periods of remission and exacerbation. The latter are consequent on physical and mental overstrain, fatigue, chilling, and are attended with intensified haemolysis and the development of jaundice. In the periods of exacerbation the patient complains of pains about the spleen and liver, tinnitus and dizziness caused by anaemia. There is also a temperature rise in this period.

Physical examination reveals a slight icteric colour of the skin and visible mucous membranes. In mild cases the patient is rather icteric than pale. The spleen and sometimes the liver are enlarged and palpable. The urine contains urobilin but no bilirubin or biliary acids. The stools are excessively coloured and hypercholic. The bilirubin content of the blood is increased to 2-3 mg% and gives an indirect reaction to diazo reagent.

Congenital haemolytic anaemia is of the hypochromic type. The peripheral blood contains microspherocytes (spherical erythrocytes and those of a smaller diameter) and reticulocytes which in certain cases reach levels of 60-90 per cent.

The disease runs a prolonged course, relapses alternating with remissions. Trophic ulcers sometimes develop on the shanks. In the presence of bilirubin stones in the bile ducts and the gallbladder attacks of cholelithiasis attended with obstructive jaundice may be observed.

Differential diagnosis is made between various forms of anaemia and Addison-Biermer's disease, aplastic and hypolastic anaemia, obstructive and parenchymatous jaundice. History taking and clinical and laboratory findings throw a clear light on the character of the disease, although certain difficulties may be encountered in differentiating it from Botkin's disease and partly from obstructive jaundice.

The *prognosis* is favourable if no pronounced jaundice is present. Ulcers of the shanks and attacks of hepatic colic make the outlook worse.

Treatment. Amelioration of the patient's condition is achieved by hygienic conditions of life and avoidance of physical and mental overstrain, fatigue and chilling. For severe anaemia iron, arsenic and cobalt preparations are administered. In the presence of progressive anaemia infarctions of the spleen, unresponsive to treatment trophic ulcers of the shanks, and frequent attacks of biliary colic exhausting the patient splenectomy is preferable. As a rule, it has a favourable effect on the patient's condition and the principal symptoms of the disease disappear or subside, although microspherocytosis persists. Consequently, a reduction in the resistance of erythrocytes and the latter's haemolysis remain. When it is impossible to excise the spleen ligation of the splenic artery and x-ray irradiation of the spleen are undertaken. These methods, however, are less effective than splenectomy.

Acquired Haemolytic Anaemia

Aetiology and pathogenesis. Acquired haemolytic anaemia (jaundice) develops in malaria, Botkin's disease, chronic sepsis, syphilis, prolonged intoxication, etc. There is an intensified decomposition of erythrocytes in the reticuloendothelial system: the spleen, bone marrow and lymph nodes.

Pathologic anatomy. There is deposition of haemosiderin in the spleen, liver and bone marrow. Infarctions and thromboses occur in the system of the portal vein and biliary thrombi in the bile capillaries are found.

Clinical picture. As distinct from congenital haemolytic anaemia this disease runs a more severe course. The patient often suffers from phlebitis and thrombosis of the splenic veins with the development of thrombophlebitic splenomegaly attended by gastrointestinal haemorrhage. In thrombophlebitis of the trunk of the portal vein ascites develops as a result of a disturbance in the portal circulation. The spleen is enlarged and firm (indurated), and in perisplenitis tender. The anaemia is more pronounced than in the congenital form of haemolytic anaemia, spherocytosis and reticulocytosis, however, being inconstant findings. There are microcytosis and leucopenia. The osmotic resistance of erythrocytes is slightly reduced, the bilirubin content of the blood is usually elevated, the reaction to diazo reagent is indirect. The level of urobilin and globulin in the urine is increased. The differential diagnosis is made between the same diseases as in congenital haemolytic anaemia. The prognosis is less favourable than that in the congenital form. Gastrointestinal haemorrhages and thromboses of the portal vein and mesenteric vessels, with a possible development of intestinal gangrene and peritonitis, are highly dangerous to life.

Treatment. The primary measure is treatment of diseases reponsible for haemolytic anaemia. Other therapeutic procedures are the same as those employed for the above-mentioned form. Splenectomy is rarely performed on patients with haemolytic anaemia. An increase in haemolysins is controlled by a prolonged administration of ACTH and steroid hormones—cortisone in a dose of 100 mg, prednisone or prednisolone in a dose of 30 mg in 24 hours. This treatment brings about a remission rather than recovery.

Anaemia Primarily Due to Intravascular Haemolysis

Anaemia caused by intravascular haemolysis may be acute or chronic. Acute anaemia occurs in intoxication with various haemolytic poisons—phosphorus, arsenic, saponins, snake venom and mushroom poison and in severe burns and transfusions of incompatible blood. Acute haemolysis is also observed in such infectious and parasitic diseases as malaria in which plasmodia produce decomposition of erythrocytes. Haemolysis is caused by the action of intravascular autohaemolysins on the blood. The following forms of acute intravascular haemolytic anaemia are distinguished: (a) children's and adolescents' and (b) anaemia of the newborn.

Anaemia in children and adolescents manifests itself suddenly and is accompanied by high fever, vomiting, symptoms of bleeding (nosebleed, cutaneous haemorrhage), enlargement of the spleen and liver. The skin and visible mucous membranes are pale and have an icteric hue. The blood exhibits hyperchromic anaemia with anisocytosis and poikilocytosis, an increased reticulocytosis, neutrophilic leucocytosis, and thrombopenia. The bilirubin content of the blood is elevated, and the urine contains urobilin.

Treatment consists in repeated transfusions of blood in doses of 250-300 ml and administration of the same remedies that are used for any form of haemolytic anaemia.

Haemolytic Anaemia (Jaundice) of the Newborn

This disease is manifested by anaemia and jaundice, enlargement of the liver and spleen. In the peripheral blood a considerable number of erythroblasts occurs. In the newborn this disease develops as a result of haemolysis if the foetus has a positive rhesus-factor, the mother having a negative one. Due to this fact agglutination and haemolysis in the newborn take place. Haemolytic anaemia develops also when the father has a positive rhesus-factor while the mother a negative. True, this combination does not necessarily produce haemolysis since haemolytic anaemia in the newborn occurs in 0.5-1.0 per cent of births, whereas marriages of rhesus-positive men to rhesus-negative women are estimated at 10-12 per cent.

Clinical picture. There are pronounced jaundice, cutaneous haemorrhages, oedema, enlargement of the spleen and liver, anaemia with a large number of normoblasts as a result of an intensified bone marrow function. The white blood presents a picture of neutrophilic leucocytosis with a shift to the left. The blood bilirubin is elevated and has an indirect reaction. The urobilin content of the urine is increased and the stools have an intense colour of bile.

The *prognosis* has largely improved after the discovery of agglutinogen (1940) and rhesus-factor identical with the factor of monkey blood (Macaca rhesus). Intrauterine and postnatal deaths are estimated to occur in not more than 10 per cent of cases.

Treatment. The newborn baby is given a transfusion of rhesus-negative blood, except for maternal blood, since the latter contains rhesus-antibodies. Exchange transfusion of blood by means of removing 300 ml of blood from the umbilical vein of the newborn and replacing it with about 400 ml of donor blood of an identical group with a negative rhesus-factor (this treatment is described in greater detail in manuals of obstetrics).

ERYTHROCYTOSIS, POLYCYTHAEMIA, ERYTHRAEMIA

Erythrocytosis is a condition when the number of erythrocytes is equal to or more than 5,000,000 in 1 cu mm of blood. Erythrocytosis results from oxygen deficiency or inspissation of blood, and may be physiologic or pathologic. The former is found during intrauterine life of the foetus when the number of erythrocytes is as large as 9,000,000-10,000,000 and is retained until the appearance of pulmonary respiration, after which the excessive erythrocytes decompose. Physiologic erythrocytosis occurs in residents of mountainous regions and is associated with a low atmospheric and partial oxygen pressure. An increase in the number of erythrocytes is noted in persons ascending to high altitudes and is attributable to oxygen deficiency resulting in erythrocytes being delivered into the blood stream from the spleen, the skin capillaries, etc.

Pathologic erythrocytosis occurs in intoxication with carbon monoxide, anilin, in hypertensive vascular disease, inspissation of blood, burns, pulmonary oedema, emaciating diarrhoea, etc. As a result of underoxygenation of the blood erythrocytosis occurs in patients with cardiovascular insufficiency, particularly in those with congenital heart disease and pulmonary stenosis.

Polycythaemia. The term polycythaemia denotes a condition in which a simultaneous increase in the number of erythrocytes, leucocytes and thrombocytes is observed. Often it is termed erythraemia, which is incorrect, since in the latter case there is an increase in the total blood volume mainly due to an increase in the number of erythrocytes.

Erythraemia is a systemic disease of the haemopoietic organs in which the erythroblastic stem of the bone marrow intensively produces erythrocytes and discharges them in excessive number into the peripheral blood.

The aetiology of this disease is unknown. Importance is attached to oxygen deficiency, a disturbance in the regulatory function of the central nervous system, influences of toxic substances. Of late, cases of transition of erythraemia into chronic myeloid leukaemia have been reported. It has been asserted on these grounds that erythraemia is a form of leukaemia. The disease afflicts persons of 40 to 50 years of age.

Pathologic anatomy. The bone marrow is crimson-red, the spleen is enlarged and contains a large number of erythrocytes and thrombocytes. The yellow bone marrow has turned red and hyperplasia of myeloid tissue is noted in the bone marrow. The visceral organs are engorged with blood.

Clinical picture. The disease in insidious in onset, tends to progress and take a chronic course. The patient complains of headaches, flushes of blood to the head, fatigability, deterioration of memory, poor eyesight, tinnitus, dyspnoea, dyspeptic symptoms and pains in the bones.

On examination the face, lips and tongue are cherry-red in colour, the scleras are injected and there is a visual disorder due to a circulatory disturbance. The spleen is enlarged, particularly in the terminal stages of the disease. The arterial pressure is in most cases within normal limits. The heart boundaries are expanded, the pulse is fast, phlebitis and sometimes symptoms of chronic nephritis being noted. The circulating blood volume is considerably enlarged.

The blood picture is of decisive importance for the diagnosis because in erythraemia the amount of haemoglobin is considerably increased being in excess of 16 g% (100 units), while the number of erythrocytes is as large as 9,000,000-10,000,000 per cu mm of blood. Examination of the blood with a haematocrit shows that dense particles constitute 70 per cent instead of the normal 45 per cent. The viscosity of the blood is increased. There is also irritation of the white stem of the bone marrow with a resulting leucocytosis of up to 30,000-40,000 per cu mm of blood and hyperthrombocytosis contributing to predisposition to thrombosis. The patient usually dies within 4-5 years from thrombosis of the cerebral or intraperitoneal vessels, displaying symptoms of fever, ascites, haematemesis, intestinal haemorrhage, and pain. If treatment is given in due time, the disease may last for decades without serious distressing symptoms, with periodic aggravations and remissions.

The *differential diagnosis* is not always simple. A similar clinical picture characteristic of erythraemia is seen with erythrocytosis. The latter is secondary to cardiopulmonary insufficiency, stenosis of the pulmonary artery, congenital heart defects, and pulmonary emphysema.

Treatment. Massive venesection (blood-letting) in quantities of 300-600 ml is undertaken once in every 5 to 8 days, sometimes in combination with oxygen therapy. Oxygen may be injected subcutaneously in doses of 300-400 cu cm (10-12 insufflations). In predisposition to thrombus formation leeches are applied to the mastoid process, particularly in combination with blood-letting. The most common is treatment with radioactive phosphorus (P^{32} per os in doses of 2 millicuries after preliminary blood-letting at one week's intervals, the total dose amounting to 6-8 millicuries). Another course of treatment is undertaken in 6-8 months. If the above-mentioned methods of therapy fail, x-ray therapy is employed in which individual bones are irradiated.

It should be borne in mind that sometimes x-ray therapy is followed by leukaemia, tumour formation, aplasia of the bone marrow. The diet must be nutritious. Restriction of the intake of meat and food containing iron is recommended.

LEUKAEMIA (LEUCOSES)

Leukaemia is a general disease of the body with a deep involvement of the haemopoietic system. It is essentially due to a rapid proliferation of rudimentary reticular cells with a disturbance in their normal differentiation and formation of pathologic foci of extramedullary (outside bone marrow) haemopoiesis in the organs and tissues which usually take no part in blood formation.

Aetiology and pathogenesis. No theory of the aetiology of leukaemia has so far received general recognition. A variety of theories have been proposed to explain its origin: (a) chemical; (b) infectious-allergic; (c) viral; (d) ionizing radiation, etc.

The chemical theory. The proponents of this theory assert that leucogenic substances (aromatic carbohydrates, benzene, various resins, anilin dyes, etc.) are formed in the body or introduced from without. It has been proved that on painting the skin with cancerogenous substances (carbon, etc.) and after the latter's subcutaneous injections experimental animals develop leukaemia. The group of endogenous leucogenic substances also include steroids bound with proteins and lipoproteins. They are also found in the sex hormones. It has been shown experimentally that leukaemia develops in mice with excised adrenal glands given injections of estrogens.

The virus theory proposed by L. Zilber is based on the fact that chickens and mice given injections of leukaemic tissue passed through filters develop leukaemia whereas filtrates from healthy tissue and organs fail to cause such changes. This theory, however, needs further substantiation.

The infectious and infectious-allergic theory proposed by D. Yanovsky has not been confirmed by epidemiologic observations. Septic processes in leukaemia, including tonsillitis, are of a secondary character.

The ionizing radiation theory. Experimental research, study of the incidence of leukaemia among roentgenologists as well as cases of leukaemia in survivors of the atomic bombing of Hiroshima and Nagasaki warrant the theory of a causal relationship between ionizing radiation and leukaemia.

The tumour theory regards leukaemia as a special form of malignant newgrowth. Cancerogenous substances sometimes produce leukaemia. Both diseases (leukaemia and malignant newgrowth) lead to hyperplasia of cells incapable of differentiation. The best therapeutic effect is produced by the same remedies: x-rays, radioactive phosphorus (P^{32}), cytostatic chemical drugs. It is beyond doubt that leukaemia and malignant neoplasms have certain features in common but it would be a mistake to regard them as being identical.

The deficiency theory. It is believed by many that a metabolic disorder in the cell nuclei is responsible for leukaemia. A great contributing role is attached to nucleic acids which regulate protein synthesis and metabolism in growing cells. It is supposed that a disturbance in nucleotide metabolism due to the influence of external and internal factors brings about hyperplasia of cells and inhibits their differentiation.

The role of hereditary factors in the development of leukaemia has been studied inadequately. It is supposed that the testes and ovaries contain the so-called leucogenic agent which may be heritable. M. Dultsin reports an observation of a family in which the father and his two sons suffered from chronic lymphatic leukaemia.

It is most probable that leukaemia is a polyaetiologic disease, i.e., it is due to various causes, both internal and external, but has a homogeneous mechanism of development, i.e., presents a monopathogenic condition. Depending on which cells predominate in the blood picture in leukaemia the latter is subdivided into the following forms: (1) reticulosis; (2) haemocytoblastoma; (3) myelosis; (4) lymphatic leukaemia; (5) polycythaemia; (6) myelofibrosis or osteomyelofibrosis. The most frequent is myeloid leukaemia, while lymphatic leukaemia is the rarest in incidence. With regard to its course leukaemia may be acute, subacute, and chronic, and for the number of erythrocytes in the peripheral blood it may be subdivided into leukaemia, subleukaemia, and aleukaemia. In certain cases, the disease process, which is aleukaemic at the outset, may develop into subleukaemia and later into leukaemia.

Leukaemia must be differentiated from a leukaemoid reaction which is associated with various diseases, such as tuberculosis of the lungs or pneumonia, and fails to progress and disappears along with the principal disease. Those who disagree that the leukaemoid reaction should be set appart from leukaemia believe that in such cases there is a reaction of a variable degree to one and the same stimulus, sometimes in transitional forms.

Acute Leukaemia (leucosis acuta)

Acute leukaemia is more frequent than other diseases of the haemopoietic system, runs a violent course, with an early disturbance in haemopoiesis, profuse bleeding, high-grade fever and often with symptoms of general sepsis.

Pathogenesis. In acute leukaemia the maturation of blood corpuscles at its earliest stages is retarded and sometimes stopped altogether. This is accompanied by a simultaneous proliferation both in the bone marrow and in various organs of haemopoietic cellular elements incapable of maturation. In this connection, undifferentiated cellular elements—haemocytoblasts, haemohistioblasts, myeloblasts, etc.—appear in the peripheral blood in addition to a considerable reduction in the number of mature and maturing cells. A marked delay in or cessation of the maturation of juvenile cells causes an absence of intermediate cells between juvenile and mature myeloid cells in the peripheral blood so that a kind of a gap appears which is termed hiatus leukaemicus.

Pathologic anatomy. Pathologic changes in the body in leukaemia vary with the period of the disease. In the initial period changes occur in the bone marrow (myeloid leukaemia) and lymph nodes (lymphatic leukaemia); in the second period, in the organs performing haemopoietic function during intrauterine life (liver, spleen, etc.); in the third period, in all organs and tissues (kidneys, lungs, heart muscle, endocrine glands, alimentary tract), where leukaemic infiltrations arise from slightly differentiated cells corresponding to the form of leukaemia. In the bone marrow of the spongy bones the cellular trabecula are thinned or disappear. Areas of necrosis and haemorrhages appear within it. The spleen and lymph nodes of the liver show myeloid infiltration and dystrophic changes. In the oral cavity, pharynx, gastrointestinal tract, and in the genital canal gangrenous-ulcerative lesions and sepsis develop, which is facilitated by a reduction in the phagocytic capacity of leucocytes. The bone marrow, spleen, and lymph nodes acquire a rusty colour due to a deposition of iron from decomposed erythrocytes. Haemorrhages occur in the skin, serous membranes, and hollow organs (stomach, intestine, kidneys, uterus, etc.) as a result of an increased capillary permeability, thrombocytopenia, and a reduction in the number of mast cells producing heparin.

Clinical picture. Acute leukaemia occurs in the following forms: (1) reticulocytosis; (2) haemocytoblastosis; (3) myeloid leukaemia.

Acute reticulosis, or systemic hyperplasia of the reticular cells, occurs in two forms. In the first one, the reticular cells are liberated from the syncytial connections and enter the peripheral blood, producing haemocytoblastosis. The second variant in which the reticular cells are absent from the peripheral blood is known as acute or subacute leukemic reticulosis.

Pathologic anatomy. There is proliferation of reticular cells in the bone marrow, lymph nodes, spleen, liver and partly in organs which do not take part in haemopoiesis (the lungs, endocrine glands, brain meninges, skin, etc.). The bone marrow of the flat bones is red or grey-red in colour. The long bones are also involved, mainly in the upper third of the femur. Microscopically the bone marrow consists of reticular cells.

Clinical picture. The disease sets in with a steep rise of temperature to 39-40°C, necrotic tonsillitis and haemorrhages in the tongue and gums. The lymph nodes, spleen and liver enlarge, and jaundice develops sometimes. In certain cases the disease process extends to other organs in which infiltrations and nodes occur, in particular in the skin and orbit, resulting in protrusion of the eyeball. The direct causes of death are cerebral haemorrhages, pneumonia, profuse bleeding, liver dystrophy, sepsis, etc.

Haemocytoblastosis runs an acute or subacute course, and is very rarely encountered after the age of 60.

Pathologic anatomy. The bone marrow is diffusely affected, dark-red and sometimes grey-green in colour, and contains haemocytoblasts, the principal maternal haemopoietic cells, which have lost their capacity for differentiation (maturation). The spleen is enlarged and filled with haemocytoblasts. The lymph nodes are enlarged.

Clinical picture. The disease is abrupt in onset, with complaints of debility, fatigability, pains in the joints, sometimes with a rise in temperature, enlargement of the lymph nodes, bleeding of the gums, and ecchymoses on the body. The patient is pale, with haemorrhagic eruptions on the skin. The spleen and liver are enlarged due to leukaemic infiltration. The latter is also observed in the lungs, pleura, bronchopulmonary lymph nodes, the acoustic nerve with the development of deafness; in the retina with a partial or total blindness; and in the kidneys with haematuria. Leukaemic infiltrations also extend to the brain and spinal cord, sometimes with haemorrhages in the vital centres of the brain. There are leukaemic pneumonia, relapse of tuberculosis of the lungs, haemorrhagic pleurisy with haemocytoblasts in the exudate sediment, etc. Catarrhal or ulcerative necrotic lesions of the mucous membranes (tonsillitis, stomatitis, gingivitis, vulvitis, etc.) are caused by a secondary septic infection and are encountered at a later time. Over the past years their incidence has declined owing to antibiotic treatment and chemotherapy.

The *blood picture* in haemocytoblastosis is highly characteristic. In bone marrow punctates the level of haemocytoblasts may reach 90-95 per cent. Sometimes myeloblasts are also discovered. Their number may, under the influence of therapy, increase considerably and even exceed that of haemocytoblasts. The number of leucocytes in the peripheral blood is increased or leucopenia is present. There is a characteristic gap (hiatus leukaemicus) between juvenile haemocytoblasts and mature cells, particularly in case of an acute course of the disease. Anaemia may at first be absent, since part of the haemocytoblasts retain their ability to maturate into erythroblasts. In addition, at the onset of the disease the blood contains a sufficient number of old erythrocytes. Later, the disease symptoms are joined by anaemia, which is due to a paralytic state of the bone marrow and profuse haemorrhages.

Acute erythraemic myelosis (Guglielmo's disease). In certain cases haemocytoblasts retain their ability to differentiate into proerythroblasts and erythroblasts. This condition was for the first time identified as an independent disease by Guglielmo (1917). This disease is encountered very rarely. In the bone marrow and the peripheral blood, in addition to haemocytoblasts, there are a considerable number of elements of the erythroid series which have lost their ability to differentiate into mature erythrocytes.

If the patient survives this period, the blood picture later becomes very typical of haemocytoblastosis because erythroblasts completely disappear from the bone marrow and the peripheral blood, while haemocytoblasts and myeloblasts increase in number. On these grounds it is asserted by many that Guglielmo's disease is simply a variety of acute leukaemia rather than an independent condition.

Acute myelosis, or acute myeloblastic leukaemia, is met with fairly often.

Pathologic anatomy. The bone marrow is succulent, dark-red in colour and consists of myeloblasts. Myeloid infiltration is also observed in the liver, spleen, kidneys, lungs, and lymph nodes. The spleen is slightly enlarged, as are the lymph nodes, the latter, however, to a smaller extent than is the case in chronic myelosis. They are of a whitish-red colour and succulent. Highly characteristic of acute myelosis are ulceronecrotic and gangrenous processes in the guns, tonsils and intestine.

Clinical picture. At the onset of the disease there are only moderate signs of general intoxication as manifested by debility, general malaise, and pains in the bones. Sometimes there is throat pain and bleeding from the gums. The patient often gives no complaints and only upon examination can one discover a slight pallor of the skin, insignificant enlargement of the cervical lymph nodes, and sometimes a positive pinch sign. In a late stage of the disease there are symptoms of progressive debility, headaches and dizziness, fever with chills, necrotic changes in the oral cavity, dispnoea, palpitation and bleeding ranging from isolated petechiae to extensive haemorrhages on the body, bleeding from the nose, genitalia, less frequently from the intestine and lungs. The spleen and liver are enlarged. The cardiac sounds are muffled. A systolic murmur is audible over the heart apex and the pulmonary artery. The pulse is frequent and the arterial pressure is reduced. In the presence of leukaemic infiltration, the lungs show signs characteristic of pneumonia. The appetite is decreased and massive gastrointestinal haemorrhages are likely to occur. The urine contains blood and casts. Cerebral haemorrhages are followed by hemiplegia and hemiparesis. There is a large loss of vision to the degree of total blindness due to retinal haemorrhage as well as deafness caused by a leukaemic infiltration of the accoustic nerve. In the bone marrow and sometimes in the periosteum there are leukaemic infiltrations which cause excruciating pains.

Blood picture. The erythrocyte count is diminished to 1,000,000-1,500,000, with a corresponding reduction in haemoglobin level (20-30 units). There is a substantial decrease in the number of reticulocytes and thrombocytes in the peripheral blood. Anaemia results from a derangement in the process of erythroblast maturation and replacement of erythropoietic tissue by leukaemic. The maturation of megakaryocytes is also disturbed. The leucocyte count is increased relatively slightly (30,000-100,000) and sometimes remains normal. Most frequently, however, leucopenia is observed. In acute myelosis the white blood almost exclusively consists of myeloblasts since the transitional forms between myeloblasts and mature leucocytes are absent, a condition known as the hiatus leukaemicus, the proportion of mature leucocytes in the peripheral blood being equal to only 1-2 per cent.

Transitional forms are sometimes observed between leucopenic and leukaemic forms of acute myelosis, leukaemia being superceded by leucopenia, or vice versa (Fig. 71).

The *prognosis* is ominous. By means of treatment acute leukaemia may be converted into a subacute form and survival time may be prolonged to 1-2 years. Death is due to general intoxication, heart failure, cerebral haemorrhage, profuse haemorrhages or secondary sepsis.

Treatment of acute leukaemia. This consists of a complex of measures. ACTH is administered intramuscularly in a dose of 20 U three times daily, while prednisone and prednisolone in a dose of 20-30 mg are given per os three times daily. These drugs are administered for 3-4 weeks. Doses must be reduced gradually. ACTH, prednisone, and prednisolone produce an antiproliferative, desensitizing, and anti-inflammatory effect and lessen the permeability of the vessel wall. 6-Mercaptopurine (6MP) is prescribed in doses of 50 mg to be taken 2-3 times daily (formula No. 139*a*). The total dose of this drug per course of treatment is up to 6-7 g. In case of haemorrhages treatment is discontinued. Highly efficacious are systematic drip transfusions of whole blood (200-225 ml), erythrocyte mass (120-150 ml) at intervals of 2-5 days. Bleeding is arrested with rutin, ascorbic acid, calcium, etc. Fever is controlled with various antibiotics. During the past few years transplantation of bone marrow compatible with respect to group and type factors has been employed.

Chronic Myeloid Leukaemia (myeloleucosis chronica)

Chronic myeloid leukaemia occurs in persons of 20 to 60, less frequently at an older age and in childhood. It is higher in incidence than lymphatic leukaemia.

In chronic myeloid leukaemia there is proliferation of myeloid tissue in the bone marrow, liver, spleen, kidneys, lymph nodes and other organs, while immature leucocytes of the myelocyte group enter the peripheral blood. Anaemia ensues since during an intensified proliferation of myeloid tissue the production of erythrocytes in the bone marrow is reduced. Haemorrhages are encountered in many organs.

Pathologic anatomy. The bone marrow of the sternum, ribs, vertebrae and of the long bones is succulent, dark-red or grey-red, sometimes with a greenish hue, and pyoid due to a large number of myeloid cells. Microscopy of tl reveals a multitude of myeloblasts, promyelocytes, cytes. The erythropoietic stem is atrophied. The sple ly enlarged and may weigh as much as 4 to 10 kg. Its s the capsule has thickened walls, with cicatricial adhesions, and visible foci of ischaemic infarctions spleen sinuses are filled with myeloid cells. The l though less than the spleen. The lymph nodes are not Myeloid infiltration is encountered also along the tract, in the kidneys, lungs, adrenals, testes, etc. The infiltrates in chronic myeloid leukaemia show a p to oxidase.

Clinical picture. The onset of this disease is in advanced cases there is debility, vertigo, fever, p bleeding, reduced working capacity, and a feeling pains in the region of the spleen. The latter is consi and sometimes extends to the small pelvis. On palp is firm and has a smooth surface. Sometimes p marked tenderness due to infarctions in the spleen tions in the surrounding tissues (perisplenitis). Ther of the liver which protrudes from under the costal n firm but slightly tender on palpation. The intestine The inguinal, axillary, cervical, mesenteric, and lymph nodes are enlarged although inconstantly a Percussion reveals tenderness of the sternum, ribs, column. In late stages of the disease there is a sev mucous membranes and skin. Leukaemic infiltrat mucous and submucous membranes of the gastr and cause dyspepsia; in the lungs they produce a of pneumonia; in the penis, a prolonged distressin pism). Sometimes the patient suffers from dyspno dilation of the heart. Symptoms of severe dystroph may be present. In relapses of the disease, the b is elevated, which is apparently due to an intensiv of leucocytes and rejuvenation of the bone marr is usually intensified. The urine contains a large a uric acid, salts of phosphoric acid, and purine bas as a result of disintegration of leucocytic nuclei. Th table, complains of a deterioration of hearing and

Changes in the blood are the principal sympton (Fig. 72). The leucocyte count is increased to 100, even more. In subleukaemic forms the number of l within limits of 20,000-30,000; in aleukaemic forn or diminished. The aleukaemic form runs an un anaemia and splenomegaly progressing slowly. The fied by needle biopsy of the bone marrow and sple

A highly characteristic sign is the appearance of immature forms of leucocytes of the myeloid group, i.e., myeloblasts, promyelocytes, and neutrophilic, basophilic and eosinophilic myelocytes. The number of juvenile, polymorphonuclear neutrophils, basophils and eosinophils is quite insignificant. The red blood shows hypochromic macrocytic anaemia, anisocytosis, and poikilocytosis.

The disease runs an undulating course with brief remissions. In relapses, deterioration in the patient's condition is accompanied by a rise in temperature, dystrophy of the liver and myocardium, thrombocytopenia, a reduction in the amount of haemoglobin, and appearance of juvenile forms of the myeloid cells. Chronic myelosis fails to respond to therapy, rapidly progresses, and terminates in death within 2-3 years. Some of its forms, however, run a very torpid course and last for 10 years and longer.

Death in chronic leukaemia is due to dystrophy of the vital organs, circulatory disorders, infarction of the spleen, profuse haemorrhage, and very rarely to a rupture of the spleen.

Chronic Lymphatic Leukaemia (lympholeucosis chronica)

Chronic lymphatic leukaemia is mainly encountered at ages of 40 to 60 years. It is manifested by enlargement of the lymph nodes, usually of the servical and axillary, but sometimes the disease is generalized in character (hyperplastic process extending to the mesenteric, mediastinal and other lymph nodes). The disease may begin in the tonsils and the salivary and lacrimal glands. Protrusion of the eyeball is encountered if a leukaemic infiltrate is present in the fundus oculi. Priapism occurs in involvement of the cavernous bodies of the penis. Mucosal haemorrhages appear in late stages of the disease and are attended with progressive thrombocytopenia. The liver is enlarged and sometimes shows dystrophic changes. Another finding, though inconstant, is splenomegaly.

The development of lymphatic infiltrates in the lungs leads to pneumonia; in the gastrointestinal tract, to gastroenteritis. Lymphatic leukaemia develops in several forms: (a) with hepato- and splenomegaly; (b) with hyperplasia of the external lymph nodes; (c) with regional hyperplasia of lymph nodes; (d) with manifest splenomegaly; (e) with an isolated lesion of the bone marrow; (f) with tumour-like proliferation of the lymph nodes in the mediastinum and the abdominal and the oral cavities.

As a rule, enlarged lymph nodes cluster but are not adherent to one another or to the adjacent organs. Microscopy reveals hyperplasia of the follicles.

Clinical picture. At an early stage of the disease the patient's general condition is satisfactory, his working capacity is retained, while the spleen, liver and lymph nodes show no marked enlargement. The patient feels no discomfort for a long time. Sometimes he is troubled only by the large size of the lymph nodes. Emaciation progresses gradually. There appear signs of a circulatory disturbance (dyspnoea, palpitation, oedema) and dysfunction of the liver due to its dystrophy. Various inflammatory processes (tonsillitis, rhinitis, etc.) are attended with a prolonged fever.

The blood picture is an important guide in the diagnosis of lymphatic leukaemia (Fig. 73). In the peripheral blood lymphocytes predominate (up to 90-95 per cent) and are seen as tiny narrow protoplasmatic forms, almost bare nuclei. Botkin-Gumprecht's shadows—cells with a loosened nucleus—are also discovered. In severe forms, prolymphocytes and lymphoblasts, and sometimes reticular cells predominate. The leucocyte count may be increased to 400,000 and more per cu mm of blood, but may also be normal (aleukaemic form). In far-advanced cases, the symptoms are joined by hypochromic macrocytic anaemia and signs of haemorrhagic diathesis (as a result of a gradual decrease in the thrombocyte count). Highly valuable for a diagnosis is needle biopsy of the bone marrow in an aleukaemic form when the lymphocyte count in the peripheral blood is not increased.

In respect of its clinical course chronic lymphatic leukaemia is divided into slowly and rapidly progressing forms. In the former case the disease lasts 8-10 years. This form is usually observed in aleukaemic and subleukaemic leukaemia.

Treatment of chronic leukaemia. Therapy consists of measures to improve the patient's general health (nutritious diet rich in vitamins, rest, etc.). Fowler's solution is administered in doses of 15 drops three times daily during a month at intervals. In case of relapse, foci of leukaemic infiltration are reduced by means of x-ray therapy and myelosan (myleran) treatment (formula No. 138*a*) which bring about a temporary relief.

Roentgen therapy consists in repeated irradiation in large doses of various parts of the spleen and bones at half-a-year intervals until the leucocyte count reaches a level of 30,000-40,000 per cu mm of blood. After the end of x-ray therapy destruction of leucocytes continues; therefore to avoid leucopenia irradiation must be stopped in due time. X-ray therapy is contraindicated for aleukaemic forms, severe anaemia, pronounced predisposition to bleeding, and cachexia.

The dose of myelosan varies with the number of leucocytes in the peripheral blood. If they number more than 200,000 the dose is 8 mg per 24 hours (to be taken in 2 mg portions four times); in a reduction to 40,000-50,000 the dose is only 2 mg per 24 hours, and in a decrease of the leucocyte count to 10,000-15,000 per cu mm myelosan treatment should be discontinued. Myelosan usually takes effect in 3 weeks' time. Maintenance doses of myelosan may be administered for a long period (1-2 years). Myelosan treatment is carried out with a check being kept on the condition of the peripheral blood.

At the onset of thrombocytopenia myelosan treatment is also discontinued and transfusion of erythrocytes and thrombocytes is prescribed.

Myelosan is contraindicated for acute and subacute leukaemia, relapses of chronic myeloid leukaemia if it runs its course after the type of acute leukaemia, also for aleukaemic and subleukaemic forms of chronic leukaemia, and severe thrombocytopenia.

Corticosteroids (prednisone and prednisolone) and vitamin B_{12} in combination with rutin (formula No. *42a*) are also used. For relapses of chronic myeloid leukaemia dopane (formula No. *140a*) is administered; in the presence of splenomegaly and in subleukaemic forms the preparation is given in doses of 6-7 g per os at intervals of 4-5 days. To avoid the risk of dyspepsia, soporifics—nembutal (pentobarbital sodium) or barbamyl (amytal sodium) (formulas Nos. 111 and 112)—are given one hour after the administration of dopane.

A leading role in the therapy of chronic leukaemia, that attended by anaemia in particular, is played by drip transfusions of blood and erythrocyte mass, as well as administration of iron preparations. Chronic lymphatic leukaemia accompanied by anaemia is treated with vitamin B_{12} given in doses of 100 γ three times a week and antianaemin given in doses of 2 ml every other day. The course of treatment lasts 1 to 3 months. Antibiotics are prescribed in secondary infection, and 6-Mercaptopurine, in a severe relapse of chronic leukaemia.

Chronic lymphatic leukaemia runs a slow course, particularly in elderly persons, without clinical manifestations. In such cases one should abstain from the use of antileukaemic remedies. Treatment with iron and ascorbic acid is preferable.

Malignant newgrowths are treated with x-rays and chemical agents of cytostatic action, ThioTEPA, in particular, in combination with blood transfusion in order to avoid anaemia. Novoembichine, degranol, and chlorbutin are also used.

AGRANULOCYTOSIS

In agranulocytosis granular leucocytes—neutrophils and eosinophils—are absent from the peripheral blood. The production of erythrocytes and blood platelets is undisturbed. Agranulocytosis is secondary to infectious diseases, in particular those caused by streptococci, and occurs also in measles, enteric fever, malaria, and staphylococcal and pneumococcal diseases. Certain drugs (salvarsan, mercury and gold preparations, pyramidon, atophan) may contribute to the development of agranulocytosis in hypersensitive subjects as well as in case of overdosage. The disease has also been traced to food poisoning, in particular after eating cereals prepared from grain left to lie in the field for the winter (alimentary aleukaemia).

Clinical picture. The disease marks its onset by a sudden rise in temperature. It is accompanied by catarrhal symptoms in the nasopharynx and the formation of necrotic ulcers in the tonsils. Identical ulcers are seen in the oral mucosa, the gastrointestinal tract, genitalia, and sometimes on the skin. Splenomegaly is frequent. The leucocyte count in this condition is markedly diminished to 1,000-500 per cu mm. An important diagnostic sign is not only a reduction in leucocytes but also complete absence of neutrophils or a diminution of their level to 2-3 per cent of the total number of leucocytes. Lymphocytes and monocytes in the blood are retained. Thus, it is only granulocytic leucocytes that completely disappear from the blood, to which fact this disease owes its name. The red blood count remains within normal limits. Haemorrhagic diathesis is absent. The disease runs an extremely acute course and in severe cases ends in death within 3-4 weeks as a result of its being joined by infection and sepsis, with symptoms of a rapid development of necrosis. When the source of agranulocystosis has been established in good time the patient may be saved by measures to eliminate the causes of the disease. A temporary amelioration is sometimes followed by a fatal relapse.

Treatment. Repeated transfusions of blood in doses of 100-200 ml are recommended. Transfusions of leucocyte mass, nucleic acid preparations (1-2 ml subcutaneously or intramuscularly) (formula No. 134) serving to intensify leucopoiesis, thesane (formulas No. 135 and 135*a*), and pentoxyl (formula No. 137*a*) are also employed.

LYMPHOGRANULOMATOSIS OR HODGKIN'S DISEASE

Aetiology and pathogenesis. The aetiology of this disease is unknown. There is a theory of its infectious origin (tuberculous, viral, etc.). A certain similarity between lymphogranulomatosis and lymphosarcoma and reticuloendothelioma has given grounds to regard it as a tumorous process. As distinct from the latter, however, lymphogranulomatosis is not accompanied by metastasization and produces only metaplasia characteristic of diseases of the haemopoietic system.

Pathologic anatomy. Lymphogranulomatosis is characterized by the presence of giant cells described by S. Berezovsky (1890) and K. Sternberg (1898). The lymph nodes are enlarged and vary in size: they contain granulomas but show no signs of caseous degeneration. Granulomas are also present in the spleen and liver.

Lymphogranulomatosis is known to develop in three stages. In the first stage there is diffuse hyperplasia of the lymph nodes which contain a large number of various cells. In the second stage reticular and plasma cells, eosinophils, and Berezovsky-Sternberg's giant cells containing several nuclei are found in the lymph nodes. In the third stage there is connective tissue growth, shrinkage of the node, and sometimes necrosis of isolated areas followed by the formation of scar tissue.

Clinical picture. The disease commonly occurs in young men. Sometimes enlargement of the lymph nodes is recognized by chance since it causes no discomfort to the patient. Later the lymph nodes involved increase in number and become firm. There is general debility, and a rise in temperature which is remittent or subfebrile. Sometimes the temperature may even be normal. Often the onset of the disease is marked by an enlargement of the lymph nodes on the neck, which is later followed by enlargement of the axillary and supraclavicular lymph nodes. In some cases the primary affection is seen in the mediastinal and mesenteric lymph nodes. A very rare occurrence is a primary lesion of the skin, usually on the back, shoulders, and thighs where firm nodules the size of a pea are found. The disease involves the lungs and pleura as a primary process or may be secondary to an extension of the disease from the mediastinum. This gives rise to wet pleurisy, the effusion containing neutrophils, lymphocytes, eosinophils, and Berezovsky-Sternberg's cells. Lymphogranulomatosis also involves the gastrointestinal tract. The oesophagus suffers less frequently. An affected stomach is ulcerated or contains mushroom-like formations attached by a broad base. Involvement of the intestine in lymphogranulomatosis is manifested by intestinal obstruction, perforation, peritonitis, and intestinal haemorrhages.

Lymphogranulomatosis produces a moderate enlargement of the spleen and liver. The findings in the nervous system are various types of neuralgia and paralyses. The blood displays hypochromic anaemia and an elevated ESR, while a relapse of the disease produces neutrophilic leucocytosis with a shift to the left to the stage of metamyelocytes and even myelocytes, and in severe cases with leucopenia. Itching of the skin is frequent.

The course of the disease varies with its form and stage. Lymphogranulomatosis occurs in acute, subacute and chronic forms, and in respect of its extension may be focal (confined to isolated foci), generalized, and cachectic.

Differential diagnosis. Needle biopsy of the lymph nodes and detection of Berezovsky-Sternberg's giant cells are decisive for a diagnosis. Lymphogranulomatosis is differentiated from tuberculosis of the lymph nodes, lymphosarcoma with metastases to the lymph nodes, reticulosis, and leukaemia. Tuberculous lymphadenitis produces suppuration of the lymph nodes with the formation of fistulas, while needle biopsy yields cheesy masses. Diagnostic puncture in lymphosarcoma produces typical cells.

The *prognosis* is unfavourable. The survival time is usually 2-4 years, and sometimes 5-6 years.

Treatment. The most common method of treating this disease is x-ray therapy by means of local or total irradiation which destroys exposed tissues. During this procedure the body temperature returns to normal, the lymph nodes diminish in size and disappear or are replaced by scar tissue. A few months later, however, another relapse occurs in which x-ray therapy is less efficacious. The latter is contraindicated in leucopenia and thrombocytopenia. The chemical remedies are novoembichine, embichine, dopane, chlorbutin, degranol, etc. These agents acting on the cell nuclei suppress glycolysis and inhibit mitoses. They only help obtain a remission lasting several months. They should not be used in anaemia, leucopenia, and thrombocytopenia. Embichine and novoembichine are administered intravenously in a dose of 4-8 mg, the total dose being 50-80 mg for a course of treatment. Dopane is given per os in the same doses. ThioTEPA is administered intravenously or intramuscularly in doses of 10-20 mg, the total dose for a course of treatment being 100-200 mg. Corticosteroids and ACTH are a valuable supplement to the above-mentioned methods of therapy. Corticosteroids are administered in large doses of 10-15 mg two or three times daily during a period of 2-3 weeks. For focal forms of lymphogranulomatosis a radical excision of the node followed by x-ray therapy may be undertaken. The diet must be nutritious and rich in vitamins.

MULTIPLE MYELOMA OR RUSTITSKY-KAHLER'S DISEASE (MYELOMATOSIS)

Multiple myeloma is a systemic disease of the haemopoietic organs and is manifested by an irresistible growth of plasma cells, sometimes with tumour-like proliferations.

Aetiology and pathogenesis. The aetiology of this disease is unknown. For its clinical course it resembles leukaemia, although it displays no characteristic changes in the peripheral blood. It also bears resemblance to a tumour, but without metastases and extension to the surrounding tissues.

Clinical picture. The patient complains of lassitude, general malaise and costal, cranial, sternal and femoral pains which grow worse on physical exertion and tapping of bones. Later bone deformities and spontaneous bone fractures may occur. Inspection reveals pallor of the skin and mucous membranes. Hypotension, a functional systolic murmur audible over the apex of the heart, and enlargement of the liver and spleen are found on examination. There is hypochromic anaemia. The white blood exhibits moderate leucocytosis, a normal thrombocyte count, and in severe cases leucopenia and thrombocytopenia. Needle biopsy of the bone marrow reveals the presence of plasma cells (in certain cases their amount is as high as 40 per cent and more) and reticular cells. Roentgenography of the affected bones shows defects of a round form ranging from several millimetres to 2-3 cm in diameter. These defects result from destruction of bone tissue and formation of plasma cells in the areas of bone death. Defects in the skull, pelvic bones, and the ribs are characteristic symptoms of the disease. Another characteristic finding in multiple myeloma is a considerable increase in the blood protein level mainly in β- and γ-globulins. The kidneys are also involved (albuminuria, cylindruria). The urine contains peculiar protein bodies known as Bence Jones protein which is precipitated from urine heated to 60°C and redissolves during further heating. Amyloidosis, renal insufficiency, and uraemia may also develop. The calcium content of the blood is in excess of 15 mg/100 ml because of bone destruction.

The *diagnosis* is established from the presence of plasma cells in the bone marrow as evidenced by a puncture from bone defects, and hyperproteinaemia with predominance of β- and γ-globulins. The disease is differentiated from cancer metastases and hypernephroma.

The *prognosis* is ominous, the disease lasting 3-5 years even if modern methods of therapy are used.

Treatment. The best effect is produced by carbolysin in combination with ACTH and corticosteroid hormones. Carbolysin is prescribed in doses of 40-50 mg to be taken once weekly (the total dose for a course of treatment is 200-250 mg). Prednisone is given in a dose of 5 mg twice daily and ACTH, in a dose of 30 U twice daily. The course of therapy is repeated after 6 months. A total of 4-6 such courses are undertaken. Other remedies which are highly efficacious sometimes are x-ray therapy, urethane in doses of 2-3 g daily, and radioactive phosphorus (P^{32}). Antianaemic treatment (blood transfusion, iron, vitamin B_{12}, liver preparations) is also indicated. The diet must be nutritious, with an adequate supply of vitamins, particularly vitamin C and those of the B-complex.

INFECTIOUS MONONUCLEOSIS OR FILATOV'S DISEASE

Infectious mononucleosis is an acute disease running its course with hyperplasia of the lymphoid apparatus, and leucocytosis with atypical lymphocytosis and especially monocytosis. This disease was for the first time described by N. Filatov.

The *aetiology* is unknown. A number of authors consider infectious mononucleosis to be a viral disease, regarding the oral cavity, pharynx, and gastrointestinal tract as the portals of entry of the infection.

Clinical picture. Infectious mononucleosis occurs in the following three forms: (a) glandular; (b) anginose; and (c) febrile.

The glandular form is most commonly found in children and adolescents, and is manifested by general debility, headaches, fever, and enlargement of the cervical, occipital, axillary and inguinal lymph nodes. The enlarged lymph nodes are firm and tender on palpation.

In the anginose form the disease begins with catarrhal or ulcerative tonsillitis, enlargement of the spleen and liver, nosebleed, and petechial eruptions on the skin.

In the febrile form there is only a slight fever attended with a mild form of tonsillitis. The diagnosis is confirmed by leucocytosis (from 15,000 to 25,000 per cu mm of blood) with atypical lymphocytes and monocytes.

Prognosis is favourable. The disease terminates in recovery within 2-3 weeks of its onset.

Differential diagnosis is made to exclude acute leukaemia, agranulocytosis, and tonsillitis.

Treatment is symptomatic. Antibiotics (penicillin, streptomycin) and of late corticosteroids are used.

HAEMORRHAGIC DIATHESIS

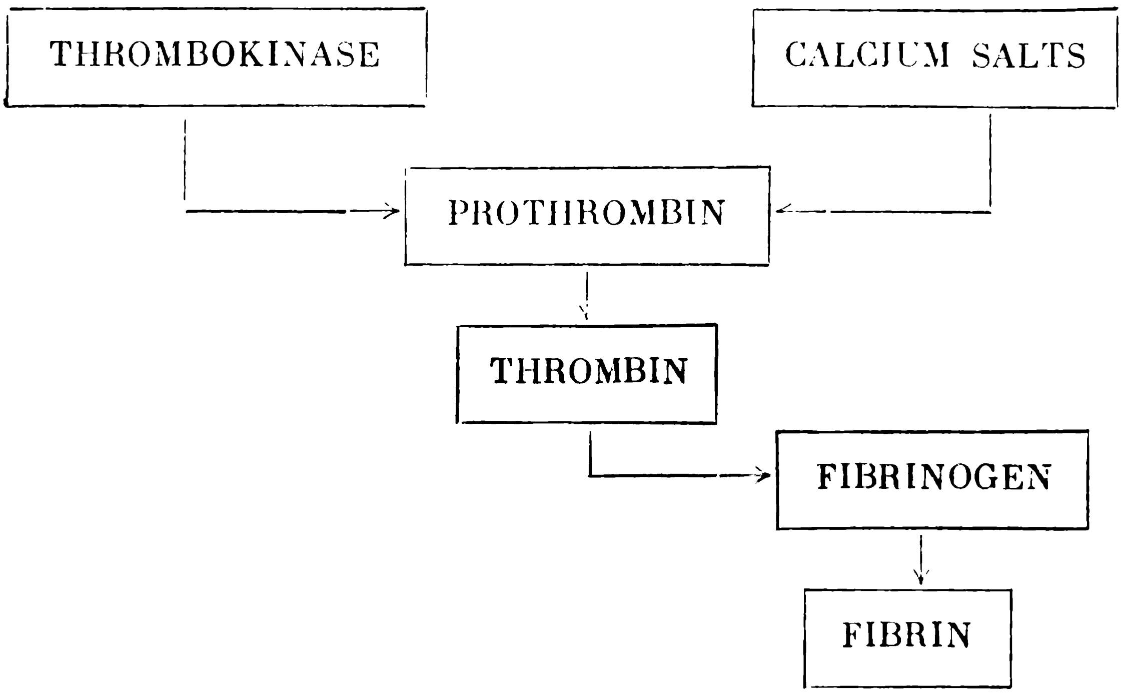
In order to gain a correct understanding of the pathogenesis of haemorrhagic diathesis and its clinical picture one should have a clear idea of the mechanism of coagulation of blood.

In healthy persons an injury to a blood vessel causes bleeding which is quickly arrested as a result of the intricate processes occurring at the site of damage. A trauma is immediately followed by constriction of the damaged vessel, which results in a lessening of bleeding. Simultaneously, blood platelets (thrombocytes) accumulate at the site of injury after which a clot of fibrin is formed stoppering the bleeding vessel like a plug (thrombosis). The process of blood coagulation itself, i.e., of fibrin clot formation is extremely intricate. For a clot to form it is necessary that the protein fibrinogen dissolved in the blood should be converted into insoluble fibrin. This conversion can take place only when fibrinogen is influenced by a special substance known as thrombin. Thrombin is not normally present in the blood, but the latter contains prothrombin from which thrombin is produced under pathologic conditions.

After a trauma the enzyme thrombokinase (thromboplastin) is liberated from the destroyed thrombocytes and tissues, owing to which prothrombin is converted into thrombin in the presence of calcium salts. Thus, the mechanism of coagulation of blood may be schematically presented as follows: under the influence of thrombokinase and calcium salts prothrombin is converted into thrombin and the latter transforms soluble fibrinogen into insoluble fibrin.

A disruption at different stages of the intricate process of blood coagulation leads to the development of various forms of haemorrhagic diathesis. For exapmle, a disturbance in thrombokinase production causes thrombocytopenic purpura. A slow-down in the conversion of prothrombin into thrombin results in haemophilia.

It should be noted that an abnormal predisposition to haemorrhages may occur not only in a disturbance in the processes of blood



coagulation but also in a pathologically increased permeability of blood vessels, for example in scurvy and haemorrhagic capillary toxicosis.

All cases of haemorrhagic diathesis may be conventionally divided into two principal groups: primary and symptomatic, or secondary.

The former includes those disseases in which predisposition to bleeding is the cardinal manifestation. The aetiology of most of them is still uncertain.

Symptomatic (secondary) haemorrhagic diathesis implies proneness to bleeding as only one of the many symptoms of a disease. This diathesis occurs in diseases of the liver, certain vitamin deficiencies (scurvy), leukaemia, etc.

In the present section only primary haemorrhagic diseases will be discussed.

Capillary Toxicosis (Schoenlein-Henoch Purpura)

Capillary toxicosis is a frequent disease characterized by a sudden eruption of haemorrhagic rashes on the skin and mucous membranes, sometimes with involvement of the joints and certain visceral organs.

The *aetiology* of this disease is uncertain. Infection, however (influenza, tonsillitis, scarlet fever, etc.), is undoubtedly a contributing factor. A certain role is played by traumas and alimentary intoxications. The capillaries are damaged and their permeability is increased. In addition, allergic changes occur in the capillaries or arterioles and venules.

Clinical picture. The principal symptom is the presence of haemorrhages in the skin and mucous membranes, joints, abdominal cavity, the kidneys, and the brain. The skin (mainly of the legs) displays punctate haemorrhages and red patches, sometimes with a simultaneous rise of temperature to 38°C and higher. In the intestinal form there are colicky pains in the abdomen, vomiting of blood, and diarrhoea with a discharge of blood. In this condition profuse haemorrhages occur in the gastric and intestinal mucosa. Sometimes necrotic sites occur in the intestine followed by ulceration, perforation, and peritonitis. In capillary toxicosis, the vessels of the brain, heart, lungs, and other organs may be involved. There is moderate anaemia, neutrophilic leucocytosis, an elevated ESR, and a positive tourniquet test.

Differential diagnosis is made to rule out dysentery, acute nephritis, rheumatic fever, sepsis, etc. The prognosis in involvement of the abdominal cavity and kidneys is serious.

Treatment. This consists in administrations of dimedrol, diazoline, rutin, ascorbic acid, calcium preparations, sodium salicylates, vitamins, ACTH, and cortisone and its derivatives.

Idiopathic Thrombocytopenic Purpura (Werlhof's Disease)

This disease is known as thrombocytopenia purpura, Werlhof's disease, benign thrombocytopenia, etc.

It essentially consists in a considerable reduction in the thrombocyte count and spontaneous bleeding and extravasations of blood.

The *aetiology* of this disease is uncertain. A contributing role is possibly played by infections and intoxications. A number of authors assert that this disease is caused by a deficiency in the body of the platelet factor influencing the maturation of the bone marrow megakaryocytes. It was supposed that thrombocytopenia results from an increased function of the spleen, which is known to be a kind of a graveyard for the erythrocytes, as it was presumably evidenced by the occurrence of splenomegaly in this disease. Splenectomy, however, failed to produce recovery. Most probably, there is a diminution in the production of megakaryocytes in the bone marrow, while the spleen inhibits their maturation.

Clinical picture. The disease is more frequent in females than in males. There are haemorrhages in the skin and mucous membranes, bleeding from the nose, gums, lungs, intestine, uterus, kidneys, and less frequently from other organs. Bleeding and extravasations of blood occur spontaneously. Later, posthaemorrhagic anaemia and splenomegaly develop. Examination of the peripheral blood reveals a marked reduction in the thrombocyte count, even to the degree of 2,000—3,000 per cu mm of blood, giant and degenerated platelets of a variable size making their appearance. The differential leucocyte count is unchanged. In the presence of a profuse haemorrhage quantitative and qualitative alterations in the red blood are noted.

In the bone marrow the number of megakaryocytes increases due to an addition of juvenile forms—megakaryoblasts and promegakaryocytes. Normal megakaryocytes account for 20 per cent of the total count. In the presence of anaemia an intensified erythropoiesis occurs in the bone marrow. Clot retraction time is increased and in periods of remission is normal. The tourniquet test (Konchalovsky—Rumpel—Leede phenomenon) is positive, the pinch symptom is also positive, the bleeding time is increased, the blood coagulation time is normal or sometimes prolonged. The disease runs a chronic course, with alternating periods of relapse and remission.

The *prognosis* is usually favourable. There is a likelihood of severe posthaemorrhagic anaemia. The working capacity is diminished.

Treatment. Bleeding is arrested with haemostatic remedies. These are calcium chloride, sodium chloride, vitamins C and K, transfusions of blood, plasma, and thrombocyte mass. Splenectomy is sometimes performed, after which the thrombocytes increase in number and the bleeding ceases. ACTH and cortisone and its derivatives are prescribed. The permeability of blood vessels is reduced with dimedrol, diazoline, vitamin P (in a dose of 0.05 g three times daily), and rutin (formula No. 42*a*).

Haemophilia

Haemophilia is a severe disease manifested by a tendency to excessive bleeding. The latter is due to a decelerated conversion of prothrombin into thrombin as a result of a dificiency of certain protein substances in the blood plasma.

This disease is hereditary, and is encountered exclusively in males. Females transmit this disease to their children, but remain unaffected themselves.

Clinical picture. The first symptoms of the disease make their appearance in childhood. Any trauma, often quite negligible (scratching or pricking), gives rise to bleeding which may last for hours and even days. Sometimes blood may escape under the skin and into the muscles and joints. In the latter case rigidity of the joints and ankylosis develop. Renal and intestinal haemorrhages are less common. Frequently recurring prolonged haemorrhages produce anaemia. Examination of the blood reveals a marked increase in the blood coagulation time. The bleeding time as well as the thrombocyte count remain normal.

Treatment. No remedy is still available to effect a complete cure of haemophilia. The most reliable method of arresting bleeding and increasing coagulation of the blood is repeated transfusion of blood or plasma in doses of 150-200 ml. Simultaneously with haemotransfusion local haemostatic remedies such as applications of cold and gauze compresses soaked in fresh blood and plasma are employed. It is not recommended to arrest bleeding by means of a tourniquet. The diet must be varied and sufficient in proteins and vitamins.

Endocrine and Metabolic Disorders

THYROTOXICOSIS OR EXOPHTHALMIC GOITRE (BASEDOW'S OR GRAVES' DISEASE)

This disease was for the first time described by Graves (1835) and later by Basedow (1840), and now it bears their names. At present it is also called thyrotoxicosis by many.

Aetiology and pathogenesis. In the majority of cases thyrotoxicosis is correlated to the effects of a severe psychic trauma, prolonged anxiety with an increased production of thyroxin, the hormone of the thyroid gland. The latter's amount is increased also due to an intensified activity of the anterior lobe of the pituitary body and its secretion of thyrotropic hormone. Thyrotoxicosis may also be caused by involvement of the thyroid gland into the infectious process in influenza, tonsillitis, malaria, rheumatic fever, syphilis, tuberculosis, etc. Apparently, the infection causes a direct damage to the gland and the central nervous system with pathologic metabolic products. A contributing role in the development of thyrotoxicosis is played by affection of other endocrine glands (sexual, pituitary, etc.).

Normally the thyroid gland consists of two lateral lobes united across the median line by an isthmus. It weighs 20 to 40 g, lies on the anterior and lateral surfaces of the larynx and trachea on a level with the sixth tracheal ring and adjoins the oesophagus. It is supplied with blood by the superior and inferior thyroid arteries which are innervated by the sympathetic and parasympathetic nervous systems.

The thyroid gland is composed of follicles—closed hollow formations of an irregular or round form which contain colloid material consisting of proteins and iodine carrying amino acids (monoiodothyrosine, iodothyrosine, thyroxin). The production and secretion of thyroxin are regulated by the central nervous system and the anterior lobe of the pituitary body which secretes thyrotropic hormone intensifying the formation of incretion. Excision of the anterior lobe of the pituitary body causes atrophy of the thyroid gland.

Thyroxin intensifies the oxidative processes occuring in the body, increases protein decomposition, oxygen consumption, elimination of water from the body, and reduces the cholesterol and fatty acid content of the blood. An increase in thyroid function is attended with an enlarged supply of thyroxin and iodine into the blood and an intensified iodine absorption by the gland. Iodine is supplied into the body with food (greens, milk, eggs, fish, etc.) and water. The normal iodine content of the thyroid gland is from one-fifth to one-tenth of the total amount of iodine in the body, whereas in thyrotoxiçosis it is increased more than ten-fold. The elimination of iodine from the body is also increased. Hypofunction of the gland entails a diminution of iodine uptake by the thyroid.

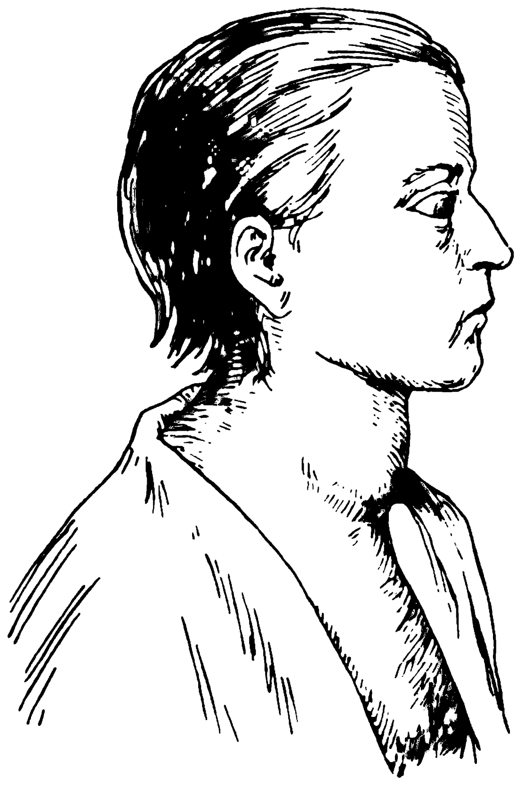


Fig. 74. Basedow's disease

Thyroid hyperfunction causes substantial changes in various organs and systems.

Clinical picture. Palpitation, early fatigue on exertion, lassitude, emaciation, irritability, insomnia, sometimes a tendency to diarrhoea are the common findings. The rate of this disease in females is 5-6 times that in males. An important symptom is enlargement of the thyroid gland which sometimes lies behind the sternum and is located by roentgenography or at operation. Retrosternal goitre causes difficulty in breathing and hoarseness.

In thyrotoxicosis there is usually a diffuse enlargement of the entire gland (Fig. 74), with a predominant enlagrement, however, of one of the lobes or the isthmus. The gland is soft in consistency, richly vascularized, and sometimes pulsating. Tremor and "gurgling of blood" are felt on palpation. Above and beside the gland there is an extensive network of skin venules which are especially engorged with blood in coughing. Vascular murmurs due to the existence of an extensive vascular network are audible on auscultation. Enlargement of the gland is known to occur in five stages: I—the gland is slightly palpable and escapes notice when the patient is swallowing; II—the gland is well palpable and noticeable in swallowing; III—the gland enlargement is readily visible; IV—the gland is markedly prominent; V—the gland enlargement is extremely severe.

The most striking findings are seen in the eyes, in particular exophthalmos, i.e., protrusion of the eyeball due to contraction of the oculomotor obliquus muscles. Severe exophthalmos may lead to ulceration and even loss of the eye as a result of the latter's inflammation. Other signs observed in addition to exophthalmos are as follows: (a) an upper lid lag on downward gaze, which is accounted for by an increased tone of the superior palpebral levator (muscle raising the upper lid), a white strip of the sclera being visible (Graefe's sign); (b) failure of convergence, i.e., loss of the capacity for fixation of the gaze upon an object placed at the nearpoint, which is due to the prevalence of the tone of the obliquus muscles over that of the internal rectus muscles effecting convergence (Moebius' sign); (c) infrequent blinking due to a difficulty in overcoming the tone of the superior palpebral levator (muscle raising the upper lid) (Stellwag's sign). These symptoms in combination with lustre of the eyes make the patient look frightened.

Other findings in thyrotoxicosis are tachycardia, left ventricular hypertrophy, an increase in the systolic and a decrease in the diastolic pressure, and murmurs over the apex of the heart and the pulmonary artery. In severe forms atrial fibrilliation without right ventricular hypertrophy is sometimes noted. The heart muscle suffers dystrophic and degenerative changes. The findings in the alimentary tract are diarrhoea, less frequently constipation, and acute paroxysmal pains in the stomach, liver and intestine. There is anacidity of the gastric juice to the degree of achylia. Acute hepatitis with jaundice may be encountered. There is a mild hypochromic anaemia, leucopenia, neutropenia, lymphocytosis, sometimes an elevated ESR, a reduction in the blood coagulation time and in blood viscosity.

In thyrotoxicosis all types of metabolic processes are pathologically intensified. There is an increase in oxygen requirements and in basal metabolism which in severe forms of thyrotoxicosis is elevated by 30-60 per cent. Thyrotoxicosis causes an increased production of cholesterol and bile acids, and a cholesterol deficiency in the liver and the adrenal glands.

The metabolic disturbance manifests itself by an increased production of urea and ammonia. Water metabolism is also disturbed, and polyuria due to a reduction in tissue hydrophilia develops. A disturbance in carbohydrate metabolism caused by the action of thyroxin on the insular apparatus is manifested by hyperglycaemia and glucosuria. Iodine metabolism is also deranged. The latter circumstance aids diagnosis when 2 millicuries of iodine-131 is injected. In thyrotoxicosis there is an intensified iodine uptake by the gland.

The intense metabolic processes lead to severe emaciation. Serious changes also occur in the neuropsychic sphere. The patient is irritable, restless and talkative, showing hyperactivity and fatigability. There is tremor of the hands, the entire body, the eyelids, and of the tongue when it is put out. The patient frequently complains of insomnia and sometimes suffers from a psychosis.

The body temperature is subfebrile and all intercurrent infectious diseases run their course with a high-grade fever. The skin is usually moist and hot, especially on the palms, and sometimes loses its natural pigment (vitiligo).

Thyrotoxicosis is attended with a functional disturbance in other endocrine glands (sexual, adrenal, pancreas). A disorder in the menstrual cycle in females and a decreased sexual desire in males are sometimes noted.

Course and complications. Thyrotoxicosis may run an acute or chronic course with periodic relapses, and occur in a mild, moderately severe, or severe form. In a mild form the pulse varies from 90 to 100 beats per min, the patient loses weight by 10-15 per cent, and his working capacity is not reduced appreciably. Moderately severe forms of the disease run their course with a partial disability, the weight is 20 per cent lower, and the pulse rate reaches 120 beats per min. Severe forms produce tachycardia, a large loss of weight, atrial fibrillation, a considerable derangement of the central nervous system, a circulatory disturbance, and complete disability. This form is likely to cause thyrotoxic crises with a severe nervous upset, hyperthermia, insomnia and coma, sometimes with a fatal outcome due to hepatic and adrenal insufficiency.

Prophylaxis and treatment. Hygienic conditions of life and measures to avoid nervous shock are recommended and working in hot shops is prohibited. Conservative and radical methods of therapy are used to reduce thyroid function. The former include treatment with iodine in small doses (formula No. 175) and diiodothyrosine (0.05 g twice daily) for a period of 20 days. The pulse rate is usually slowed down and basal metabolism reduced. Upon the appearance of coryza, palpitation, and diarrhoea (iodism) the administration of these drugs is discontinued.

The patient's condition is also considerably improved by means of therapy with 6-methylthiouracil. At first the drug is administered in a dose of 0.2 g three times daily until the symptoms of thyrotoxicosis have abated, then in a dose of 0.2 g twice daily, and later in a dose of 0.1 g until a complete cessation of symptoms (usually in 1-2 months'time). This is followed by maintenance treatment in doses of 0.1-0.05 g in 24 hours during a period of 4-6 months. With a reduction in the number of leucocytes to 4,000 and granulocytes to 30 per cent this treatment is discontinued and thesane (formula No. 135 and 135*a*) is prescribed. This drug may be taken in a dose of 0.01-0.02 g 3-4 times daily or as a 1 per cent solution administered intramuscularly in a dose of 1-2 ml 3-4 times daily. Another remedy is pentoxyl (formula No. 137*a*) to be taken in a dose of 0.2-0.3 g 3-4 times daily at meals.

Mercasolil (methimazole) (formula No. 177*a*) is administered per os. Its dosage varies with the gravity of the symptoms of thyrotoxicosis: in mild and moderately severe forms the dose is 0.005 g to be taken 2-3 times daily after a meal, in severe forms, 0.005 g 3-4 times daily. Treatment with this drug is continued for a long time (up to several months) until a stable therapeutic effect is obtained. It is contraindicated in the same cases as methylthiouracil: nodular forms of thyrotoxicosis, leucopenia, diseases of the liver, and pregnancy.

Treatment with iodine-131 has become fairly common. It is based on the fact that in thyrotoxicosis iodine uptake by the thyroid gland is increased. Upon injection of radioiodine in a dose of 2-4 millicuries, which may be increased to 13 millicuries whenever required, an iodine reserve (depot) is formed in the thyroid gland and the latter becomes exposed to internal irradiation inhibiting its function. Radioiodine may cause hypothyrosis, leucopenia, thrombopenia; it is contraindicated in pregnancy and lactation.

Digitalis treatment is undertaken in atrial fibrillation and circulatory insufficiency. Luminal is prescribed for an intense excitement and insomnia.

The diet must be nutritious and rich in carbohydrates. The intake of proteins is limited because of their stimulating properties.

Thyroidectomy is indicated when conservative therapy has proved a failure, and is performed after a preliminary course of conservative treatment. When surgery is impossible x-ray therapy may be helpful.

MYXOEDEMA

This disease was for the first time described by Gull (1873) as a diffuse mucous oedema of the skin. Later it was named myxoedema, cachexia strumipriva. Myxoedema is a rare disease. It is commonly encountered in obliterated forms (hypothyrosis), mainly in women during the climacteric.

Aetiology and pathogenesis. It has been established today that myxoedema and its obliterated forms occur in experimental animals after a total resection of the thyroid gland and also, although extremely rarely, after surgery for thyrotoxicosis. In addition, myxoedema may develop after acute (influenza, rheumatic fever) and chronic (tuberculosis, syphilis, etc.) infections, exposure of the gland to x-rays, psychic trauma, and lesions of the anterior lobe of the pituitary body (adenohypophysis) causing a deficiency of thyrotropic hormone. In children myxoedema results from underdevelopment of the gland or infection and manifests itself after the child has been weaned and thereby deprived of its supply of thyroidin with maternal milk.

All these facts are evidence in favour of myxoedema developing as a result of total or partial failure of thyroid function, which is indirectly confirmed by a considerable diminution of the amount of iodine in the gland.

Pathologic anatomy. There is underdevelopment or fibrous degeneration of the thyroid gland and atheromatous changes in the blood vessels.

Clinical picture. The patient complains of easy fatigability, pains in the joints and muscles and increased sensitivity to cold. There is a slowing of physical and mental activity, apathy, loss of memory, disinclination to talk, somnolence, and frequent depression (Fig. 75). The face is mask-like and puffy, particularly about the eyes and neck. There is a pad-like mucous oedema in the subclavian spaces. Cutaneous oedema extends to the subcutaneous cellular tissue. The lips are thickened. The skin is dry because of a reduced secretion of the sudoriferous glands, oedematous and thickened, with poor sensitivity and pallor. The hair is brittle, the eyelashes as well as hairs on the pubis, in the armpits and, in men, on the face come out. The nails are fragile, the teeth crumble and fall out, and the gums bleed.

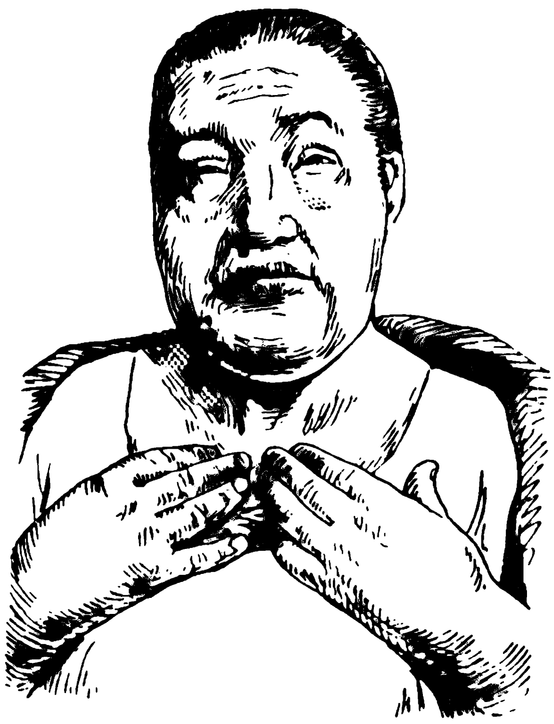


Fig. 75. Myxoedema

The respiratory rate is equal to 8-10 respirations per minute. The voice is coarse due to oedema of the larynx and vocal cords. The pulse is slowed down to 40-50 beats per min, the boundaries of the heart and the atrioventricular bundle are expanded, the cardiac sounds are muffled, the heart contractions are weak and superficial, atheromatous changes being present in the aorta and coronary vessels. There is a scanty discharge of urine with a high specific gravity and a small content of common salt, apparently due to an absent regulating role of thyroxin in water-salt interchange. A decrease in appetite, stubborn constipation and bloating of the abdomen are noted.

The principal clinical manifestation of myxoedema is a general metabolic disorder. Basal metabolism is always below normal or close to the lowest level. As a result, the body temperature is considerably reduced, while infections run their course only with a subfebrile temperature.

There is a disturbance in fat metabolism, with obesity and hypercholesterolaemia. The assimilation of carbohydrates is considerably intensified, glycogen production is increased, while the sugar content of the blood ranges from 70 to 80 mg/100 ml. The latter changes are accounted for by a reduction or complete absence of the inhibitory action of thyroxin on insulin secretion. The amount of iodine both in the thyroid gland and the blood is diminished, as is also the iodine uptake of the gland. Sexual desire is decreased, and there is sterility in women. The blood exhibits hypochromic anaemia with leucopenia and lymphocytosis.

Congenital myxoedema runs its course with certain characteristic manifestations. Children are dwarfish and slow in learning to walk, while the eruption of the teeth and closure of the fontanelles are delayed. The face is puffy, the tongue thickened and protruding from the mouth, the voice is hoarse, and a "potbelly" (large abdomen) develops. The sexual glands, the external genitalia are underdeveloped, while the secondary sex characters (hairiness, mammary glands) fail to develop at all. Sometimes such children become idiots.

In acquired myxoedema the physical and mental development proceeds normally until the onset of the disease, after which no further growth is noted. Timely identification and treatment of hypothyroidism forestall the development of severe forms of the disease and in certain cases contribute to complete recovery of the thyroid function.

Myxoedema and hypothyroidism must be differentiated from diseases of the kidneys and heart which run their course with considerable oedema. Renal diseases are excluded when protein, blood and casts are absent from the urine. Heart diseases are characterized by cyanosis, tachycardia, erythrocytosis, and electrocardiographic alterations. In many cases decisive diagnostic findings are a reduction in basal metabolism and a favourable effect produced on the course of the disease by thyroidin therapy. Myxoedema differs from hypophyseal dwarfishness in that in the latter case the mental ability develops quite normally. Endemic cretinism is very similar to myxoedema since it also results from thyroid hypofunction due to a fibrocystic process and destruction of the thyroid parenchyma. It is encountered exclusively in regions where endemic goitre is prevalent.

Treatment and prophylaxis. Prophylaxis consists in controlling acute and chronic infections, and a sparing attitude to the thyroid gland during surgery and radiotherapy.

Thyroidin treatment (formula No. 162) has produced brilliant results: the patient's activity increases and somnolence disappears, with recovery of the normal cardiovascular function and diminution of the cardiac silhouette. Thyroidin is administered in a dose of 0.1 g 2-3 times daily. After an increase in basal metabolic rate, which takes place not earlier than within 1-2 months of the beginning of therapy, smaller doses are given at intervals for a long period of time. Upon termination of therapy all symptoms of myxoedema recur.

A highly important measure is specific therapy in tuberculous (chemical agents, antibiotics) and syphilitic (salvarsan, bioquinol) lesions of the thyroid gland, if the infection has not produced irreversible fibrous changes in the latter. Transplantation of an animal or human thyroid gland into the abdominal cavity under the skin, on the chest and neck has proved futile because former symptoms reappear after the resolution of the transplant. Parathyreocrine (parathyroidin) (formula No. 163) in a dose of 0.1 g 3-5 times daily, as well as ovaricrine (folliculin) are recommended in order to recover the function of the sexual glands.

ENDEMIC GOITRE (STRUMA ENDEMICA)

Endemic goitre is encountered fairly often and is widespread in high mountainous areas, swampy and forested regions, often with a sandy soil.

In the black-earth belt endemic goitre is almost absent. The best-known areas where goitre is endemic are in Switzerland, the Carpathian Mountains, Africa, Australia, America (upper reaches of the Mississippi River), near the spurs of the Himalayan Mountains. In the USSR endemic goitre was especially common in the Western Ukraine, Byelorussia and Karelia, the Mari Autonomous Republic, in certain regions of Central Asia, the Altai Mountains, the Baikal littoral. Georgia and Upper Svanetia.

Aetiology and pathogenesis. Endemic goitre is associated with inadequate iodine reserves in the external environment. This is proved by the following facts: (a) goitre is not prevalent in regions where natural iodine supply is sufficient; (b) it occurs after a relative reduction in iodine intake; (c) it is cured by iodine preparations. The iodine content of food varies with its amount in water and the air. Iodine is especially plentiful in sea weeds and fish, while in fresh-water plants and animals its quantity is lower. The amount of iodine in the external environment is gradually decreased in proportion to the altitude of a locality above sea level. The higher the locality the less its natural iodine reserves.

The theory of goitre genesis from iodine insufficiency is also confirmed by an increased iodine uptake of the thyroid gland in healthy persons inhabiting endemic goitre foci as has been shown in investigations using iodine-131. In addition, goitre patients have a considerably reduced iodine level both in the blood and the thyroid gland.

In each endemic focus perfectly healthy persons are also met with. Apparently, for a goitre to develop other conditions are necessary in addition to iodine deficiency. Among them are unsanitary housing and living conditions, malnutrition and inadequate water supply.

Goitre most commonly occurs at ages of 10-20 years, less frequently after 30 years. Sometimes it develops in the newborn, females being more susceptible than males, apparently because of the specific features of hormonal function in women, a change in the nervous system during the menses and pregnancy. Heredity plays no contributing role as is evidenced of by children growing and developing without any symptoms of goitre after their parents have moved to a region free from a goitre epidemic or after a prophylactic administration of iodine.

Pathologic anatomy. Goitre may be nodular, diffuse or mixed, with symptoms of hyperplasia of glandular tissue and increase in colloid material and, on the contrary, with atrophy of glandular tissue. In mountainous areas nodular goitre is more common. Goitre may be bilateral or unilateral. The size of goitre is determined according to the five degrees of its development (see "Thyrotoxicosis", p. 528).

Clinical picture. The majority of goitre patients present no complaints. The cardinal sign of goitre is enlargement of the thyroid gland which sometimes grows to a huge size and causes a respiratory obstruction and difficulty in deglutition. In such cases dry cough and a changed voice may be present. Sometimes a diffuse enlargement of the thyroid gland occurring at puberty or in menstruation is of a temporary character and does not lead to the formation of goitre.

Goitre may be soft, somewhat dense and firm in consistency, readily movable, immobile with regard to the trachea and spinal column, or limited in mobility. Sometimes, the thyroid gland embraces the trachea with its lateral lobes. Goitre may be retrosternal, retrotracheal and laryngeal. Vascular murmurs may be audible over the gland. The subcutaneous veins at this site are dilated owing to a hindered drainage of blood through the deeper veins. A circulatory disturbance causes a pressure rise in the pulmonary circuit, right ventricular hypertrophy and distention.

In time the development of goitre usually ends in hypothyroidism (myxoedema), in the most severe cases symptoms of endemic cretinism make their appearance; in rare instances there is transition to thyrotoxicosis.

Complications. Endemic goitre may produce the following complications: cerebral haemorrhages, inflammations of the thyroid gland (strumitis), malignant neoplasms, compression of the adjacent organs. Malignant degeneration of goitre causes an intensified growth of the gland with its considerable induration and limitation of its mobility. Malignant neoplasms are more often encountered with nodular goitre.

Prophylaxis and treatment. The principal prophylactic measure is administration of iodine in endemic areas, beginning from infancy. This prevents goitre or checks its progression in children. In the USSR factory-made iodized common salt containing 25 g of potassium iodide per kg is used. This has brought about a steep reduction of goitre incidence.

In endemic areas an addition of iodized salt to farm animal feeds (silage) produces a healthful effect both on animals and man, in the latter case through ingestion of milk and meat. The diet must be diversified and rich in vitamins. Cod liver oil containing much iodine is recommended. For symptoms of hypothyroidism thyroidin in doses of 0.1-0.3 g is prescribed. Special attention should be paid to improving housing standards, water supply, sewerage, the development of horticulture and vegetable growing and normalizing the dietary patterns. With a failure in conservative treatment and the onset of secondary thyrotoxicosis as well as in mechanical compression of vital organs surgery is required.

HYPOPARATHYROIDISM OR PARATHYROID TETANY

In man there are two pairs of parathyroid glands (epithelial bodies), totalling 0.12-0.25 g in weight. They secrete parathyroid hormone regulating calcium metabolism. Calcium is contained in bones, all organs and tissues. It increases the tone of the nervous and vascular systems, reduces the permeability of cellular membranes. Calcium is supplied to the body with food, is absorbed in the small intestine with bile acids in the presence of vitamin D, and is eliminated from the body by the mucosa of the large intestine (colon mucosa) and partly with urine. The calcium content of the blood ranges from 9 to 12 mg/100 ml. With parathyroid hormone deficiency the amount of calcium in the blood is diminished (hypocalcaemia). Functional insufficiency of the parathyroid glands causes accumulation in the blood of guanidine and histamine which stimulate the endings of the motor nerves thereby causing tetanic convulsions.

Hypoparathyroidism is characterized by spasm of both the striated and smooth muscles.

Aetiology and pathogenesis. Hypoparathyreosis more frequently occurs after an excision of the parathyroid glands during an operation on the thyroid gland. A lesion of the parathyroid glands may be produced by an inflammation, phlegmons or traumas to the neck, etc.

The pathogenesis of hypoparathyreosis essentially consists in parathyroid hormone deficiency which in addition to convulsions causes a metabolic disorder, mainly in calcium, carbohydrate and partly protein metabolism, as is evidenced by the presence of guanidine and histamine in the urine. Poor assimilation of calcium and diminution of its amount in the blood increases the excitability of the entire nervous system, its parasympathetic part in particular, and the muscular system. As a result, any stimulation of the nerve trunks produces tetanic convulsions.

Pathologic anatomy. Autopsy often reveals atrophied parathyroid glands, sometimes haemorrhages and ulcerations in the gastrointestinal tract, liver, and kidneys occurring as a result of toxaemia.

Clinical picture. The patient's complaints are a tingling and creeping sensation and periodic painful spasms of many muscle groups. As a rule, convulsions set in suddenly as a result of mechanical, chemical and psychic stimulations, recur several times a day and continue for hours. The convulsions begin in the upper and lower extremities and extend to the torso and face. The hand assumes a characteristic position known as an obstetrician's hand. In spasm of the lower extremities the shape of the foot resembles that of a horsehoof (talipes equinus). Spasm involves the masticatory muscles (trismus) and the oral muscles. Spasm of the facial muscles causes a permanent sardonic smile. Children often develop a dangerous laryngeal spasm (stridor).

There is pallor, fragility of the nails, brittleness of hairs which tend to come out, and many carious teeth. Adults are liable to paroxysms of respiratory failure. The arteries are rigid. During an attack the urine is alkaline, and contains histamine, guanidine and methylguanidine, which is evidence of a severe disturbance in hepatic function. The calcium content of the blood is reduced to 4-6 mg/100 ml, the amount of phosphorus and potassium is increased, particularly during a paroxysm.

Certain symptoms may be evoked artificially. Among them are: (a) Trousseau's sign—obstetrician's hand—appearing if the circulation to the arm is occluded by a tourniquet; (b) Chvostek's sign—a quick contraction of the muscles of the eye, face, wings of the nose, and corners of the mouth in response to slight tapping with a plexor over the facial nerve in front of the tragus of the ear. These signs are quite striking during a fit of convulsions, whereas in periods of remission they are much less severe or absent altogether.

The disease runs a chronic course. Exacerbations commonly occur in spring, in the period of hormonal shifts within the body, and partly because of depletion of vitamin D reserves, as well as with diarrhoea and infections.

A number of diseases also run their course with tetany: among them are spasmophilia in children, intestinal diseases (enteritis, sprue), infectious diseases, poisoning with carbon dioxide, lead and ergot. It is encountered in pregnancy and lactation, with stenosis of the pylorus attended with gastroectasis. These conditions can be differentiated from hypoparathyroidism by taking note of the fact that in the latter case a diminution of the calcium content of the blood is due to a lesion of the parathyroid glands, whereas in other diseases the amount of calcium is within normal limits, save for spasmophilia in which, in addition to hypocalcaemia, guanidine is found in the urine.

The prognosis is hopeful in respect of survival but the patient is tormented by excruciating pains.

Treatment. Subcutaneous and intramuscular injections of parathyroidin in doses of 1-2 ml 2-3 times daily are recommended until the calcium content of the blood is brought up to 12 mg/100 ml (calcium level must be determined every 10-12 days). In case of a reduction in calcium below 10 mg/100 ml treatment is resumed.

Simultaneously with parathyroidin 5-10 ml of 10 per cent calcium chloride is injected intravenously or one tablespoonful of its 10 per cent solution is given per os three times daily; calcium gluconate is administered in doses of 2-6 tablets 2-3 times daily, bromide and tincture of valerian being prescribed to reduce nervous excitability.

BRONZED SKIN DISEASE OR ADDISON'S DISEASE (MORBUS ADDISONI)

The adrenal glands lie above the kidneys, are enclosed in a connective tissue capsule, and consist of two layers—an external cortical layer occupying four-fifths of the adrenal gland, and an internal, medullary layer. Their average weight is 7.5 g. Adrenalin is produced in the medullary layer. It increases the vascular tone, accelerates cardiac contractions, relaxes the bronchial musculature, elevates the basal metabolic rate, promotes the concentration of liver glycogen and increases the sugar content of the blood. The adrenal cortex secretes a large quantity of corticosteroids which are subdivided into glucocorticoids, mineralocorticoids and ketosteroids. Under the influence of glucocorticoids (corticosterone, 11-dehydrocorticosterone, 17-oxyhydrocorticosterone) glycogen deposition in the liver and protein disintegration are increased. Mineralocorticoids (aldosterone) retain sodium and water in the body and contribute to an intensified potassium excretion. Ketosteroids regulate sexual function.

The activity of the adrenal glands is controlled by the higher parts of the central nervous system through the adrenocorticotropic hormone of the pituitary body. Experimental excision of the adrenal glands causes the animal's death within a few days. The life of such animals is saved by transplantation of the cortical rather than medullary layer.

Bronzed skin disease which was first described by Addison (1855) usually results from a tuberculous (caseous), less often syphilitic and very rarely sclerotic, lesion of the adrenal cortex. This causes atrophy of the adrenals and a steep reduction in the secretion of corticosteroids. Not infrequently a reduction in cortical function is noted after certain diseases—influenza, tonsillitis, erysipelas, malaria.

Clinical picture. Addison's disease occurs in both sexes, usually at a young age. The patient complains of easy fatigability, insomnia, depression, sacral pains, loss of appetite (anorexia), weight loss, and impotence. These complaints are accounted for by a sharply reduced production of cortical hormones, which leads to glycogen depletion in the liver and muscles, sugar (glucose) deficiency in tissues, dehydration of the body (aldosterone) and impotence (corticosterone). The skin becomes bronze-coloured all over as a result of deposition of the pigment melanin. Pigmentation is particularly striking in parts of the body exposed to light and friction by clothes and in areas normally pigmented, i.e., the areolas about the nipples, skin folds on the genitalia, the forehead, places exposed to pressure from the belt, garters, and also on postoperative scars. A bronze darkening first appears on the mucous membrane of the lips, cheeks and hard palate. Other important symptoms of Addison's disease are a considerable reduction in the cholesterol and sugar content of the blood as well as hypotension. A reduction in maximum arterial pressure is caused by weakening of the heart muscle due to its glycogen depletion, and that in minimum arterial pressure, by a diminution in the amount of desoxycorticosterone.

Other prominent clinical symptoms of Addison's disease are gastrointestinal disorders: anorexia (loss of appetite), belching, heartburn, nausea, vomiting, periodic epigastrial pains, achylia, constipation, sometimes a catarrhal condition in the intestine with exhausting diarrhoea. These disturbances are apparently caused by trophic disorders in the vessels and by a deranged mineral metabolism.

Glycogen depletion in the liver as well as the latter's intensified fat supply and consumption, dehydration of the body and anorexia result in severe emaciation.

The neuropsychic sphere exhibits a variety of disturbances, such as an easy excitability, headaches, dizziness, insomnia, hallucinations, delirium, which result from an inadequate blood supply to the brain and its malnutrition. The body temperature is below normal while fever is encountered with tuberculous intoxications and thermal neuroses. Severe dehydration of the body may give rise to polyglobulia and erythrocytosis. The leucocyte count is normal or, in case of infection, increased. The number of lymphocytes and eosinophils is enlarged. The sodium, chlorine and cholesterol content of the blood is reduced while the potassium level is elevated.

Addison's disease may run its course in a severe, moderately severe, or mild form, and in respect of duration it may be acute, subacute, or chronic. Prior to the advent of adrenocortical hormone therapy the survival time in this disease did not exceed 2-3 years. The application of hormonal as well as antituberculous drugs has considerably prolonged the life of patients suffering from Addison's disease. Fulminating forms ending in death within a few hours of onset are also encountered, and chronic forms sometimes take an acute course with a sudden death due to a cardiovascular or cerebral circulatory failure.

Addison's disease has to be differentiated from pellagra, haemochromatosis (bronzed diabetes), scleroderma, cancerous, tuberculous cachexia, pigmentation in pregnancy, Basedow's disease, etc. Pellagra is characterized by deposition of pigment on the hands and exposed parts of the body, diarrhoea and dementia. Haemochromatosis causes enlargement of the liver and spleen, diabetes mellitus, etc.

Prognosis. The outlook of Addison's disease has largely improved owing to the advances achieved in the specific therapy of tuberculosis and adrenal hormone replacement therapy. The patient, however, should avoid traumas and infections which aggravate the course of the disease because of a marked depletion of the glycogen reserves in the body.

Treatment. Complete bed rest is recommended. Patients with a mild form and some with a moderately severe form of the disease must avoid mental and physical overstrain. They are allowed to perform only their habitual and easy work with a systematic application of hormone and other therapy. Specific therapy (neosalvarsan, bioguinol, streptomycin, etc.) is used for Addison's disease of tuberculous or syphilitic aetiology. The diet must be low in potassium and contain a considerable amount of carbohydrates which are ingested several times daily in order to replenish the glycogen reserves. The common salt intake should not be less than 15-20 g in 24 hours. Cortin, an adrenocortical extract, is also prescribed to prevent the loss of sodium with urine. It is injected intravenously every 4-6 hours in combination with a 5 per cent common salt solution and glucose. Intramuscular injections of cortisone in a dose of 20-15 mg daily as well as desoxycorticosterone in a dose of 5, 10, or 20 mg are recommended. In addition, glucose with ascorbic acid in a dose of 100-200 mg is administered intravenously. During the past few years desoxycorticosterone has been used in the form of crystals which are injected subcutaneously in order to form a reserve (depot). This obviates the need for injections during a period of several months.

DISEASES OF THE PITUITARY BODY

The pituitary body is oval, weighs 0.5 g and lies on the sphenoid bone of the skull in the depression of the Turkish saddle (sella turcica). It consists of two main lobes—the anterior (glandular) and the posterior, and the intermediate part (pars intermedia hypophyseos). The posterior lobe communicates with the 3rd ventricle. The pituitary body is supplied with blood through the internal carotid arteries. It is innervated by the carotid plexus.

The pituitary body as an appendage of the brain is an intermediate link in the regulation of endocrinic and metabolic processes in the body. Its hormones influence the functions of the brain, the higher and vegetative centres, but the brain, in turn, also regulates the activity of the pituitary body.

The anterior lobe of the pituitary body secretes a hormone controlling the growth and weight of the body (growth hormone). Excision of the anterior lobe of the pituitary body inhibits the growth of animals. It is supposed that the growth hormone is produced by the eosinophilic cells of the anterior lobe. The basophilic cells of this lobe produce two hormones controlling the growth of the seminal vesicles, the prostate and sperm production. Transplantation of the pituitary body promotes the growth of the ovaries, genital organs in sexually immature females of mice and rats. Excision of the pituitary body in adult animals causes atrophy of the genital organs. The thyrotropic hormone secreted by the anterior lobe contributes to thyroxin synthesis and its secretion into the blood stream.

There is an association between the pituitary body and the adrenal glands. An adrenocorticotropic hormone acting on the adrenal cortex has been isolated from the basophilic cells of the anterior lobe. In the intermediate lobe a melanophoric hormone is produced. A corresponding preparation, known as intermedin, is highly efficacious in pigmentary retinitis, vitiligo, etc. The posterior lobe of the pituitary body secretes three hormones: (a) vasopressin, which causes constriction of arterioles, elevates the arterial pressure and increases the heart's action; (b) antidiuretic hormone inhibiting the excretion of urine from the body; (c) oxytocin which intensifies contraction of the smooth musculature of the uterus, facilitates labour and contributes to quick separation and expulsion of the placenta. The posterior pituitary hormones are present in the medicinal preparation pituitrin P, which is used in hypotension, for intensifying uterine function in labour, in weakness of the intestine, and in diabetes insipidus, because it contains antidiuretic hormone.

DIABETES INSIPIDUS

Diabetes insipidus (Greek *diabetes*—a siphon, *insipidus*—tasteless) is a symptom complex characterized by the passage of large quantities of pale urine of low specific gravity and by intense thirst.

Aetiology and pathogenesis. The disease is essentially associated with hypofunction of the posterior pituitary regulating water-salt metabolism, and may be due to tumours of the pituitary body and the diencephalon (cancer metastases, adenoma, sarcoma, glioma, etc.), a syphilitic process in the diencephalopituitary region, malaria, typhoids, whooping cough, epidemic encephalitis, basilar meningitis, scarlet fever, pneumonia, trauma, and severe emotional disturbances. Signs of diabetes insipidus sometimes appear in pregnancy and disappear after labour.

In the pathogenesis of the disease a role is played by an inadequate production of antidiuretic hormone which normally controls absorption in the renal tubules of a considerable part of water and, along with the latter, common salt. A deficiency of this hormone produces a disturbance in the concentrating function of the kidneys, so that polyuria becomes necessary in order to excrete wastes from the body. This entails a considerable loss of liquid from the body (dehydration), dryness of the skin and intense thirst.

Clinical picture. The cardinal symptoms are polyuria and insatiable thirst. The patient frequently passes large portions of urine of low specific gravity (1.001-1.005) and drinks an average of 10 to 15 litres of water daily, preferring cold tap or spring water. When he is unable to drink water in quantity, the loss of urine persists all the same, which results in severe dehydration, the thirst becoming so intense as to make the patient drink his own urine. The thirst is particularly insatiable after ingestion of salty food.

There is dryness of the skin, general debility, loss of weight, headaches, depression and impotence. The cardiovascular system and the kidneys are unaffected, and the arterial pressure is not elevated. Changes in the Turkish saddle (sella turcica) are recognized by x-rays only in the presence of a pituitary tumour. In this case the patient complains of headaches, vomiting, and a visual disorder, if the tumour is exerting pressure on the chiasm. Simultaneously, an increase in the intracranial pressure is noted.

Diabetes insipidus must be differentiated from certain diseases running their course with polyuria: diabetes mellitus, polyuria of renal origin, neuroses. In polyuria due to chronic nephritis the urine contains protein and erythrocytes, as distinct from the urine of diabetes insipidus, while its specific gravity never falls to such a low level as in the latter's disease. When the patient is deprived of water, the specific gravity of urine is not increased either in granular kidney or in diabetes insipidus since in both cases liquid absorption in the tubular apparatus is impaired. After injection of pituitrin P, however, the specific gravity of urine in diabetes insipidus is considerably increased as distinct from the condition prevailing in granular kidney. In diabetes mellitus the urine contains sugar, and its specific gravity is above normal. In neuroses with polyuria water deprivation results in a rapid elevation of the specific gravity of urine, whereas patients with diabetes insipidus tolerate this test poorly, the urine specific gravity remaining unchanged.

Course. Diabetes insipidus caused by organic changes in the pituitary body tends to progress rapidly and ends in death if surgery is impossible. With a syphilitic aetiology, however, recovery may be brought about by specific therapy. Diabetes insipidus of traumatic origin may end in spontaneous recovery as a result of a gradual resorption of haemorrhagic exudate in the diencephalopituitary region.

Prophylaxis and treatment. It is necessary to avoid emotional upsets and physical traumas, and undertake an early treatment of syphilis. Treatment consists in a systematic replacement therapy with pituitrin P administered once or twice daily in a dose of one ampule subcutaneously or in the form of a snuff—aduirecrine (in a dose of 0.05 g 2-3 times daily), which is active during 12 hours. Transplantation of the pituitary body eliminating pathologic symptoms for a week or even several months has also been used. Sedatives (bromide, tincture of valerian) are also recommended. The intake of common salt must be strictly limited.

ACROMEGALY (ACROMEGALIA)

Acromegaly (Gr. *akron*—extremity, *megalos*—large) is a diseased condition characterized by overgrowth of the extremities. It results from eosinophilic adenoma of the anterior lobe of the pituitary body and an increased production of growth hormone, as well as the growth of a tumour in the diencephalon or a lesion of the diencephalon and the pituitary body due to a congenital or acquired syphilis. A contributing role is also played by pregnancy and physical trauma. The disease is encountered at ages of 20 to 40 years, mainly in women, rarely in adolescents and children.

Clinical picture. Acromegaly is manifested by enlargement of the extremities, the phalanges, thickening of the cranial bones, hypertrophy of the skin, visceral organs and atrophy of the internal genital organs (gonadal atrophy). The nose, ears, head and lips are enlarged, the superciliary arches and cheekbones are prominent. There is prognathism, the teeth are widely spaced, the tongue is so enlarged as to fill the entire oral cavity and interfere with speech. Because of the thickening of the vocal cords, the voice is hoarse and deep, particularly in women. The hands and feet are enlarged not only in length but also in width and are spade-shaped (Fig. 76). There is an intensified growth of the clavicles, sternum and ribs, enlargement of the anterior part of the thorax with symptoms of kyphosis of the cervicospinal region and lordosis of the lumbar parts. The hairs are thickened, and the skin is dry, wrinkled and thickened. The heart, liver, kidneys, spleen and intestine are noticeably enlarged (splanchnomegaly). There is enlargement of the mammary glands and nipples in women and in men, with secretion of colostrum. The skeletal muscles are thickened and a growth of physical strength is noted, which is later followed by muscular weakness. In a full-blown disease the patient complains of headaches and dizziness (vertigo) caused by a rise in the intracranial pressure due to a tumour. There is apathy and somnolence. Sometimes the patient complains of a visual disturbance resulting from pressure exerted by a pituitary tumour on the optic chiasm, if the tumour extends from the Turkish saddle and grows in the direction of the chiasm (blindness on the temporal side of the visual field—temporal hemianopsia).

In acromegaly disturbances also occur in other endocrine glands, in particular thyrotoxicosis develops resulting from an increased production of thyrotropic hormone regulating thyroxin formation and secretion into the blood. Signs of diabetes mellitus are also noted. Apparently the growth hormone of the pituitary body incites the insular apparatus of the pancreas to an active state, eventually leading to its emaciation. There is also a change in gonadal function. In the majority of cases sexual function is decreased, with cessation of the menses and development of sterility in women and a markedly reduced libido or total impotence in men. These changes in the geni-

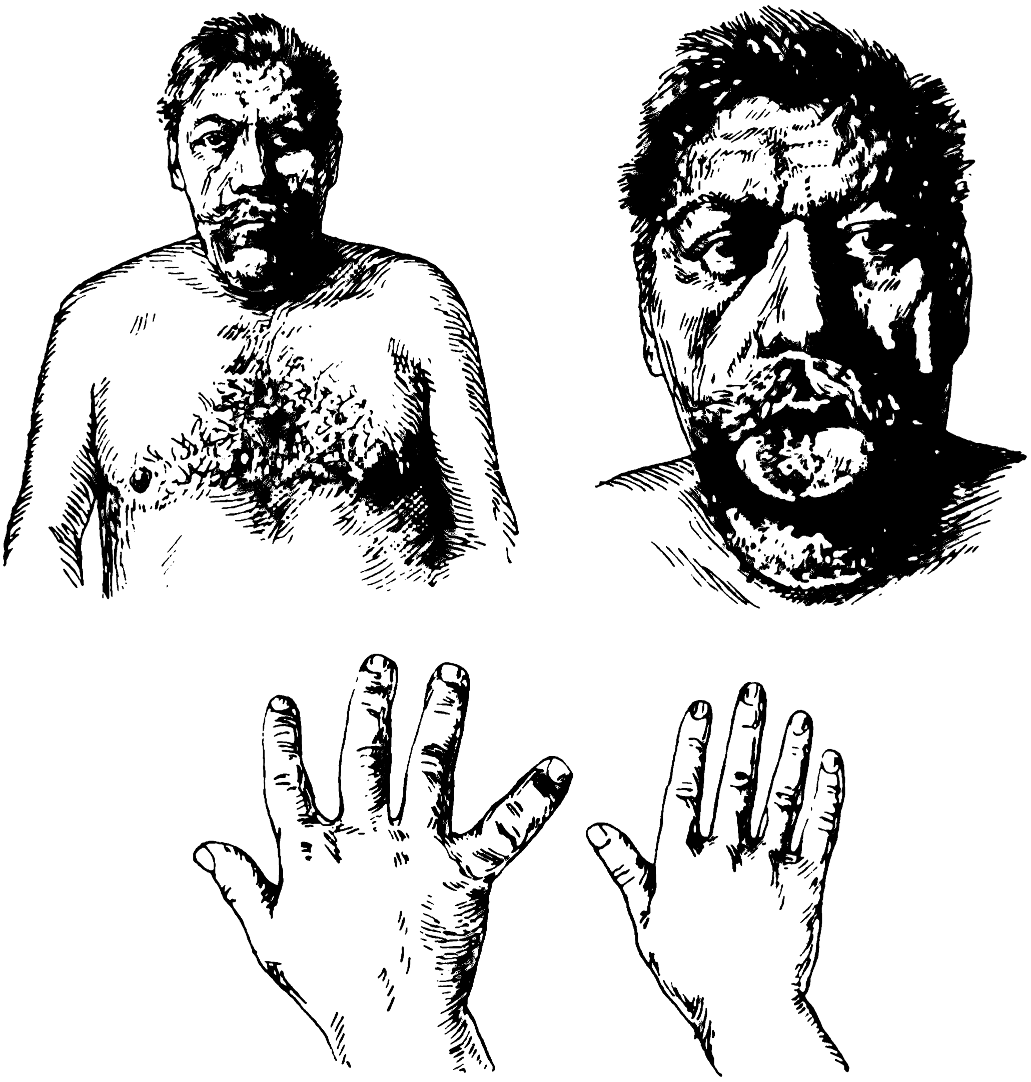


Fig. 76. Above: acromegaly; below at left: the hand of an acromegalic; at right: the hand of a healthy person

tal sphere are accounted for by gonadotropic hormone deficiency. In women there is an excessive growth of hair (virilism) and the development of male secondary sex characters—moustache and beard. Virilism results from an increased secretion by the anterior pituitary of adrenocorticotropic hormone which stimulates adrenocortical function.

The diagnosis of acromegaly is confirmed by x-rays which show enlargement of the bones as well as of the Turkish saddle with dilatation of its entrance if the disease has been caused by a tumour.

Prognosis. The outlook of acromegaly varies with the degree and direction of tumour growth, rapidity of the development of local and general cerebral symptoms, the degree of elevation of the intracranial pressure, a metabolic disorder and the state of the cardiovascular system.

Sometimes the growth of a tumour looks like being stopped, the disease running an extremely slow course, the patient retaining his working capacity for a long period.

Death is usually due to the following causes: purulent meningoencephalitis, infections (tuberculosis, pneumonia, etc.), diabetes mellitus with a comatose outcome, cardiac failure, general marasmus with total blindness and a rise in the intracranial pressure.

Treatment. A suspected pituitary tumour is treated by surgery which sometimes produces favourable results. This is indicated, above all, in cases of a fulminating course of acromegaly, with a rapid loss of vision and progression of other symptoms and in a failure of x-ray therapy. The latter, however, may be efficacious in which case the menses reappear, headaches cease, as does the growth of the extremities, and the narrowed field of vision is expanded. Acromegaly of syphilitic origin requires specific therapy (arsenic, bioquinol), and sexual hypofunction is treated with sex hormones. Otherwise, treatment is symptomatic.

DIABETES MELLITUS

This disease takes its name from the Greek words *diabetes*—siphon and *mellitus*—honey, sweet. Diabetes mellitus is caused by a disturbance in the neuroendocrine system and a general but mostly carbohydrate metabolic disorder.

Mering and Minkowski produced a clinical picture of diabetes mellitus by means of excision of the pancreas in dogs (1889). The Russian physician L. Sobolev proved that the islets of Langerhans secrete an antidiabetic hormone. It was only 20 years later, in 1921, however, that Banting and Best succeeded in isolating this hormone from the pancreas. They named it insulin. Later it was found that the islets which were first described by Langerhans in 1869 consist of heterogeneous groups of cells: beta-cells producing insulin, alpha-cells secreting lipocaine (lipocaic) which plays an important role in lipoid metabolism, as well as glucagon which inhibits insulin production.

Aetiology and pathogenesis. Diabetes mellitus essentially consists in inability of the body to deposit and consume glucose (glycogenogenesis and glycogenolysis respectively). This results in unused sugar being accumulated in the blood (hyperglycaemia) and excreted with urine (glucosuria). A disturbance in carbohydrate metabolism is due to inadequate insulin production by beta-cells.

Diabetes mellitus occurs due to various environmental influences. Important pathogenetic factors are: (a) a disorder in the neuropsychic sphere (fright, death of the patient's near relatives, prolonged emotional upset, etc.); (b) obesity resulting from habitual overeating of foods rich in saccharine substances; (c) a sedentary way of life; (d) toxic affection of the insular apparatus after acute (influenza, tonsillitis, typhoid, German measles) and chronic (tuberculosis, syphilis) infections. In certain patients diabetes mellitus develops in the presence of organic lesions of the anterior lobe of the pituitary body (acromegaly, Itsenko-Cushing's disease, etc.). Diabetes mellitus sometimes results from an anatomical lesion of the insular apparatus (atrophy, inflammation, fatty infiltration, fibrosis, hyalinosis, etc.) due to Botkin's disease (infectious hepatitis), cholelithiasis, hypertensive vascular disease, atheromatosis, pancreatitis, cancer of the pancreas, etc.

Diabetes mellitus sometimes develops in persons with a normal production of insulin by the pancreas. In such cases insulin is destroyed by the enzyme insulinase which is intensively secreted in persons with a liver disease. In addition, the death of hepatic cells liberates a proteolytic enzyme inactivating insulin.

The frequency of diabetes mellitus among members of one and the same family is accounted for not only by a congenital defect in the insular apparatus but also by identical conditions of life.

Carbohydrate metabolism is also regulated by the pituitary body, adrenal glands and thyroid. An increased hormonal activity of the anterior lobe of the pituitary body inhibits the function of the insular apparatus. In Itsenko-Cushing's disease an excessive secretion of adrenocorticotropic hormone (ACTH) intensifies the production of the adrenal hormones—glucocorticoids. An increased production of adrenalin by the adrenal glands causes hyperstimulation of the sympathetic nervous system resulting in an increased supply of glucose into the blood stream, with hyperglycaemia and glucosuria. The mechanism of the development of hyperglycaemia and glucosuria is possibly the same in psychic trauma and in cerebral haemorrhages. In thyrotoxicosis the development of diabetes mellitus is caused by the inhibitory effect of thyroxin on the insular apparatus.

Insulin deficiency leads to a disturbance in the deposition of glucose in the liver and muscles, an increased production of new glucose in the liver and inhibition of glucose consumption in tissues. These processes are responsible for hyperglycaemia.

In a healthy person the kidney filtrate usually contains the same amount of glucose as the arterial blood. Sugar, however, fails to be eliminated with urine, because it is fully reabsorbed in the tubules. When the sugar content of the blood is in excess of 140-160 mg/100 ml, the glomerular filtrate contains a considerably larger amount of sugar and is partly excreted with urine.

Hyperglycaemia results in dehydration of tissues since glucose excretion with urine involves a loss of liquid (polyuria), which may be as large as 3-10 litres in 24 hours. Dehydration entails an intense thirst—polydipsia.

In case of a considerable loss of sugar with urine and insufficient energy derived from sugar consumption the body supports its vital processes by spending energy produced by an increased decomposition of proteins and fats. Not only food but also organic and skeletal muscle proteins are consumed in considerably larger quantities.

An intensified decomposition of fatty tissue results in an increased amount of fat in the blood—lipaemia. The level of fat in the blood is equal to 5-15 per cent instead of the normal 1 per cent. The cholesterol content of the blood is elevated, cholesterol being deposited in the skin in the form of nodules (xanthomatosis) and cholesterol ester in the arterial walls. An increased consumption of fats results in the production of a considerable amount of fatty acids—stearic, palmitic, oleic. The latter are normally oxidated, just as carbohydrates, to carbon dioxide and water, but in diabetes mellitus this process is arrested at the stage of formation of acetone bodies—betaoxybutyric, oxybutyric acid and acetone—because of a disturbance in carbohydrate metabolism. Thus, fats as well as proteins cause ketonaemia and ketonuria when consumed incompletely.

An affection of the nervous system occurring in diabetes mellitus and manifested by neuralgia, neuritis, etc., is most probably a sequel to B_1 avitaminosis present in diabetics because of their diet being deficient in foods rich in this vitamin (cereals).

Pathologic anatomy. In the majority of cases atrophy, lipomatosis and sclerotic changes are found in the pancreas. The number of the islets of Langerhans is decreased by more than 75 per cent. The weight of an atrophied gland is less than 80 g. Sometimes no changes are found in the islets. There is a deposition of glycogen in the epithelium of the renal tubules, whereas its amount in the liver is extremely meagre. A disorder in lipoid metabolism results in fatty degeneration of the liver.

Clinical picture. The principal symptoms of diabetes mellitus are polyuria, intense thirst (polydipsia), and an increased appetite (polyphagia). The amount of urine passed in 24 hours is sometimes as large as 10 litres. The urine is pale, contains sugar in tracer quantities or as much as 14 per cent, and is of high specific gravity (1.030 and higher). These symptoms result from hyperglycaemia and glucosuria. The presence of sugar in the urine without hyperglycaemia is uncharacteristic of diabetes mellitus, whereas in genuine diabetes running its course with hyperglycaemia glucosuria may be absent because of a disturbance in sugar filtration by the renal tubules when the latter are contracted.

The patient's weight may over a relatively short period reduce by 10-12 kg, with a simultaneous development of severe general debility. Weight loss and debility are due to a disorder in the carbohydrate consumption of tissues, an increased decomposition of proteins, fats, accumulation of toxic ketone bodies in the blood, and the development of acidosis.

Diabetes mellitus causes a number of substantial changes in organs and tissues. The facial vessels are dilated, the patient's complexion being pale pink. In certain persons the soles and palms are stained yellow as a result of a disturbance in the normal production of vitamin A from the provitamin carotin and deposition of lipochrome in tissues. In such cases the intake of foods containing carotin (butter, carrots, egg yolks, etc.) must be limited. Dehydration and disturbance of the normal nutrition of the skin causes the development of furuncles, carbuncles and phlegmons, erythematosis, sepsis. Often, there is itching of the skin with scratching and pyodermia because of an increased irritability of the sensory nerve endings and, in women, itching in the perineal region due to the presence in the urine of sugar, which is a favourable medium for fungal flora.

Persons with diabetes mellitus develop atheromatosis of the coronary and cerebral vessels and of the aorta, angina pectoris and myocardial infarction, atherosclerotic cardiosclerosis and myocardial dystrophy. Among the less frequent changes is a circulatory disturbance in the lower extremities caused by atherosclerosis of the larger arteries and calcinosis of the muscular membrane with the appearance of the so-called intermittent limping. At the same time the patient often complains of pain in the gastrocnemius muscles felt in walking. Sometimes there is dry or, in case of an intercurrent infection, moist gangrene of the toes and fingers with a further extension of the gangrenous area.

The tone of the heart muscle falls because of its glycogen depletion, the pulse is quickened upon overexertion, breathlessness and oedema of the lower extremities making their appearance. The respiratory organs exhibit pharyngitis, laryngitis, bronchitis, pneumonia, and pulmonary tuberculosis associated with dehydration of the body and a reduction of its resistance to infection. Changes in the alimentary tract are also noted. Stomatitis, glossitis, pyorrhoea, gingivitis and dental caries are frequent. There is a reduction in stomach secretion to the degree of achylia. The liver is infiltrated with fat, sometimes considerably enlarged, and in severe cases of diabetes even cirrhosis of the liver with splenomegaly may develop. Diabetes mellitus is sometimes concurrent with cholelithiasis due to hypercholesterolaemia and a disturbance in the normal correlation between cholesterol and bile acids. Sometimes degenerative changes occur in the tubular apparatus of the kidneys, with albuminuria, less frequently cylindruria, as well as inflammatory changes associated with the growth of bacterial flora in a favourable sugar medium. The most frequent complications in the eyes are iritis and cataracts. Substantial changes are also noted in the neuropsychic sphere. The patient often complains of headaches, dizziness (vertigo) and disturbances in sensation. There is liability to neurosis and neuralgia of a variable localization.

Course and complications. Diabetes mellitus may run its course in a latent, mild, moderately severe or severe form.

The latent form shows no clinical symptoms. Glucosuria and hyperglycaemia manifest themselves only after an excessive intake of carbohydrates. With a well-balanced diet these symptoms are absent; they may be revealed, however, by a test with a sugar load of 50-100 g. At the same time a high sugar content of the blood, a prolonged (over 3 hours) persistence of blood sugar at a high level and its slow reversal to the initial level are also noted.

The mild form is characterized by a normal working capacity if the patient is maintained on a physiologic diet without insulin. Hyperglycaemia does not exceed 200 mg/100 ml, and the amount of sugar in the urine in 24 hours is not more than 5 per cent of the sugar value of the food intake in 24 hours (the sugar value in 24 hours is equivalent to the sum total of carbohydrates and 50 per cent of proteins). Ketonuria and ketonaemia are absent.

In a moderately severe diabetes mellitus in a patient maintained on a physiologic diet without insulin the working capacity is reduced. Hyperglycaemia is within limits of 200-300 mg/100 ml. At times hyperketonaemia and ketonuria are notable. The patient requires a moderate quantity of insulin.

The severe form of diabetes mellitus is characterized by substantial hyperglycaemia (300-400 mg/100 ml), an increase in the amount of sugar in the urine to 30-50 per cent in 24 hours, in the 24 hours' sugar value, permanent hyperketonaemia, ketonuria, a periodic precomatose state. The patient is disabled and requires a considerable amount of insulin. The most formidable complication of diabetes mellitus is diabetic coma caused by a lesion of the central nervous system with ketone bodies. It is preceded by a number of precursory symptoms—intensified polyuria, thirst, debility, somnolence, nausea, vomiting, loss of appetite, headaches and dizziness. In the precomatose period there is an increase in the sugar content of the blood as well as the urine; in addition, ketonuria makes its appearance because of the presence of acetone, aceto-acetic and beta-oxybutyric acids. If no emergency aid is given in this period, the onset of coma is inevitable. The latter is characterized at first by an intense excitement, insomnia, a noisy (Kussmaul's) or periodic (Cheyne-Stokes's) respiration, followed by a state of depression, somnolence, and total unconsciousness. Such patients usually have a pale face, with sharp features, a small and frequent pulse, the body temperature and arterial pressure being below normal, while the eyeballs are hypotonic and pit under pressure applied to them. An acetone odour is smelt in the air exhaled, and the urine discharge is markedly diminished. In coma, hyperglycaemia attains a level of 400 mg/100 ml and higher, with a high-degree glucosuria, a steep fall in reserve alkalinity (below 30 vol.%), and the development of acidosis.

Differential diagnosis. In diabetes insipidus polyuria and polydipsia may be observed, while the specific gravity of urine is not high, and glucosuria and hyperglycaemia are absent. In renal diabetes the sugar load causes an increase in the sugar content of the urine without hyperglycaemia. Diabetes mellitus must be differentiated from pancreatic symptomatic diabetes due to syphilitic, cirrhotic, purulent pancreatitis, when in addition to glucosuria symptoms characteristic of a disturbance in pancreatic digestion—pains, diarrhoea, jaundice, a disorder in enzyme production, etc.—are noted. In bronzed diabetes (haemochromatosis), besides glucosuria and hyperglycaemia, there is a characteristic colour of the skin and cirrhotic changes in the liver, spleen, etc.

Prognosis is favourable in the absence of complications and if treatment is correct. In untreated and inadequately treated cases death usually results from complications—myocardial infarction, gangrene of the extremities, etc.

Treatment and prophylaxis. It is necessary to eliminate factors likely to cause diabetes mellitus (nervous shocks, overeating, sedentary way of life, etc.). Prior to the advent of insulin therapy diabetes mellitus was treated by maintaining the patient on a diet of a low caloric value with a restricted intake of carbohydrates and proteins 50 per cent of which are converted into carbohydrates. Certain authors recommended intakes of fat (up to 200 g and more in 24 hours). Fasting days were also prescribed sometimes.

Fasting days and a nonphysiologic diet reduced the sugar content of the blood but simultaneously debilitated the body, impaired its resistance to infection, hyperketonaemia often developing after habitual eating of fat.

Under the present conditions the patient may be maintained on a physiologic diet if insulin is available. Its caloric value must be varied from 30 to 70 Cal per kg of body weight with the character of a particular job (at rest 30 Cal, for light work 40 Cal, for moderately difficult work 50 Cal, and for hard physical work 60-70 Cal). The daily diet must contain 60 per cent carbohydrates, 15 per cent proteins and 25 per cent fats. For example, in a diet of 2,800 Cal prescribed for a patient weighing 70 kg carbohydrates account for 1,680 Cal, proteins for 420 Cal, and fats for 700 Cal. Since 1 g of carbohydrates and 1 g of proteins produce an average of 4 Cal each and 1 g of fat 9 Cal the patient must be given 420 g of carbohydrates, 105 g of proteins, and 77 g of fat. Food products must be selected in account with the need for supply of vitamins A and those of the B and C complexes. Two lumps of sugar may be given as part of the carbohydrate intake. If during physiologic diet therapy the sugar content of the urine does not exceed 5 per cent of the total sugar intake one should abstain from insulin injections. The following principles should be followed in insulin therapy: (a) precautions should be taken against complete elimination of sugar from the urine to avoid a hypoglycaemic state; (b) due regard must be paid for the fact that one unit of insulin may reduce the urine sugar from 1 to 5 g; (c) if the insulin dose is insufficient for bringing the sugar content of the urine to 5 per cent of the total sugar intake in 24 hours, this dose may be increased consecutively at a rate of 1 unit of insulin per 4-3-2 g of carbohydrates. An example follows. The urine voided contains 60 g of sugar, the total sugar intake in 24 hours is 350 g. Upon deduction of 5 per cent from urine sugar which will make 17.5 g, the insulin dose must be calculated to reduce urine sugar by 42.5 g. The first dose of insulin amounts to only 8 units.

Should this dose prove insufficient it is gradually increased in order to reduce urine sugar to 5 per cent of the 24-hr sugar intake.

The 24-hr insulin dose, if smaller than 25 units, is administered in a single injection half-an-hour before meals, whereas that larger than 25 units is given in two or three injections at intervals of 6-8 hours, i.e., when the action of insulin is reduced. Food rich in carbohydrates is given within half-an-hour and within 3-4 hours of insulin injection when its action attains a maximum. In order to obviate the need for multiple insulin injections, a new preparation known as protamine-zinc-insulin (combination of insulin with zinc and protein), whose action grows during the first 12 hours and decreases during the following 12 hours, has been suggested. After an overnight administration of zinc-insulin the intake of carbohydrates is begun in the morning.

Over the past few years sulpha drugs—nadisan, invenol, oranil, Rastinon (tolbutamide), etc., which lower the sugar level, have been increasingly used in clinical practice. These drugs are quickly absorbed, retain their concentration in the blood for a long time, and are eliminated from the body with urine at a rate not exceeding 20 per cent daily. Usually, 4 g (8 tablets of 0.5 g) are taken after meals on the first day, 3 g on the second day, 2 g on the third day, and 1 g from the fourth day onwards until a stable effect is achieved, after which treatment with the drug is discontinued. The after-effects of these drugs last for days, less frequently for weeks. They may be administered in combination with insulin. It should be borne in mind at the same time that 1 g of these drugs is equivalent to 10 units of insulin. These drugs are contraindicated for a severe diabetes mellitus, infections, kidney and liver diseases, and leucopenia. In such cases, as well as in complications of diabetes, insulin therapy is necessary.

Total absence or insufficiency of lipocaine leads to a fatty infiltration of cells, ketonaemia and ketonuria. In such cases insulin is of little effect, therefore it is administered in combination with lipocaine in a dose of 0.2-0.3 g 2-3 times daily. Lipocaine contributes to oxidation of fat in the liver and its elimination from the latter. For pregnant women with diabetes the insulin dose should be maintained to avoid emaciation of the foetal insular apparatus.

Diabetic patients should strictly observe the rules of personal hygiene (rest, light exercise or physical work) and are advised to take treatment at a health resort.

DIABETIC COMA

The precursors of diabetic coma are general debility, decreased appetite, headaches, pains in the extremities, muscular weakness, sometimes dyspeptic symptoms (constipation, diarrhoea, vomiting, abdominal pains, tension of the abdominal wall, etc.). Coma not recognized in good time and consequently untreated may prove fatal.

A patient with diabetic coma is motionless, the skin is hyperaemic, less frequently dry, the skin turgor is reduced, the lips and tongue are dry. The patient's consciousness is dimmed, and a noisy Kussmaul's respiration is noted which in severe cases develops into Cheyne-Stokes respiration. There is somnolence which in the absence of medical assistance progresses to sopor and later to a deep coma.

The air exhaled smells of acetone (rotten apples). The pulse is frequent and small, the arterial pressure is reduced. The muscular tone is markedly diminished, the eyeballs becoming soft and pliant under pressure because of dehydration of the body. The tendon reflexes are markedly diminished and convulsions are sometimes seen. The body temperature is reduced or elevated in case of an infection antecedent to coma. The blood and urine sugar levels are considerably elevated, the urine voided contains ketone bodies, as well as protein, erythrocytes, tube casts, neutrophilic leucocytosis with a shift to the left being present in the peripheral blood.

When protein, erythrocytes and tube casts are present in the urine a differential diagnosis has to be made between diabetic and uraemic coma; evidence in favour of the former is the presence of sugar in the urine, its high specific gravity and absence of hypertension.

An immediate subcutaneous injection of 50-100 units of insulin is indicated. In severe cases, when a collapse is attended with a disturbance in absorption insulin in the same dose is injected intravenously. Subsequently, insulin is administered in a half-dose (25-50 units) every 2-3 hours until the cessation of coma, after which 10-15 units of insulin are injected subcutaneously every six hours. In coma the administration of protamine-zinc-insulin as well as nadisan, Rastinon, etc., is contraindicated. When using insulin in large doses hypoglycaemia is prevented by an intravenous injection, simultaneous with insulin, of 20 ml of a 40 per cent glucose solution, preferably within an hour of the first insulin injection. If the patient is able to swallow, intravenous glucose injections may be replaced by sweetened tea and fruit water. To eliminate dehydration of the body and intensify the excretion of ketone bodies, 1 to 2 litres of physiologic solution must be administered subcutaneously or by a drip infusion. To compensate for the alkaline loss one litre of a 2 per cent sodium bicarbonate solution is injected by a drip enema. An intravenous infusion of a 10-20 per cent common salt solution in a dose of 40 ml is also indicated since in ketonaemia the body loses its sodium and chlorine reserves. Upon liquidation of diabetic coma the patient is enjoined to avoid vigorous movements and abstain from meat and fatty foods during a few days. Later conventional insulin therapy is instituted gradually.

HYPOGLYCAEMIC COMA

Hypoglycaemia usually occurs due to insulin overdosage. Should medical aid be unavailable the disease may end in death. Hypoglycaemic coma is characterized by a sudden feeling of hunger, irritability, lassitude and palpitation. In certain patients coma is attended with headaches, tremor, diplopia (double vision) and profuse sweat. The gaze is fixed, and movements are constrained. Sometimes hypoglycaemia is accompanied by hallucinations and changes in the cardiovascular system. The arterial pressure is reduced and the pulse is frequent. The sugar content of the blood is decreased.

The diagnosis of hypoglycaemia presents no difficulty. The patient usually suffers from diabetes mellitus and receives regular insulin injections. Difficulties may be encountered when a patient is admitted in an unconscious state and without a case history. The diagnosis becomes clear, however, upon examination of the blood for sugar.

In difficult cases the differential diagnosis must be made between hypoglycaemia and diabetic coma. The former sets in suddenly, sometimes within several minutes, with a feeling of intense hunger, but without thirst. There is diplopia, moist skin, frequent profuse perspiration, facial pallor, increased muscular tone, intensified tendon reflexes, tremor and convulsive twitchings, and a normal ocular tension.

It is mandatory to give the patient readily assimilated carbohydrates: 30-50 g of sugar, jam, honey, candies and pastries if the patient is in a conscious state. When peroral administration is impossible 30-50 ml of a 20 or 40 per cent glucose solution is injected intravenously. Also helpful is injection of 1 ml of a 0.1 per cent adrenalin solution which rapidly elevates the blood sugar level. The result may sometimes be immediate, and symptoms of coma vanish. Hypoglycaemic coma mistaken for diabetic coma may be treated with insulin and is likely to end in death.

Besides hypoglycaemia caused by insulin overdosage the so-called spontaneous hypoglycaemia due to a tumour or hyperplasia of the pancreatic islets is also known. This condition is termed hyperinsulinism—an excessive insulin secretion. Hypoglycaemia unconnected with hyperinsulinism may also occur. For example, there is recurrent hypoglycaemia when the liver is incapable of storing sufficient glycogen (in cirrhosis). Attacks of hypoglycaemia during persistent vomiting in pregnancy are also due to a decreased level of glycogen in the liver.

The hypoglycaemic syndrome may often be present after a surgical operation on the stomach. In lesions of the central nervous system hypoglycaemia is also possible. In hyperinsulinism associated with

OBESITY (ADIPOSITAS SEU OBESITAS)

Accumulation of fat in the body beyond the physiologically normal limits is known as obesity. Ecxessive deposition of fat may be an independent disease or a symptom of some other ailment. Obesity is a widespread condition. Some cases of a colossal weight gain are known. For example, a certain Edward Bridge who died at the age of 30 weighed 308 kg. Fieldmarshal Mathias Gallas had such a huge belly that he had to carry it on a special cart. Edward Rostan of New York weighed 268 kg and, being unable to move about, spent all his life sitting and looking out of the window.

Aetiology and pathogenesis. Obesity occurs when the caloric intake exceeds the energy requirements of the body; however, cases of obesity being absent in persons prone to overeating are also frequent. In addition to overeating an important aetiologic factor in obesity is a metabolic disorder. It may be concluded on these grounds that the causes of obesity may be external (overeating) and internal (metabolic disorder).

Among the external causes an excessive intake of food, above all carbohydrates, is important. This is especially common among cooks, confectioners, as well as persons prone to abuse of alcohol—vodka, wines, etc. One gram of alcohol contains 7 Calories.

Habits for overeating may be implanted at an early age as a result of poor guidance, overfeeding children after an infection when their reflexes to plentiful food are fortified. Overeating is also associated with an increased appetite regulated by the central nervous system.

Among the external causes responsible for obesity a sedentary way of life, absence of exercise as well as neglect of sports and physical culture are important.

Of the internal factors contributing to an excessive weight gain a reduced basal metabolic rate and a disturbance in endocrine function should be noted.

Obesity is often seen in the period of decrease in sexual function: in women during the menopause, after a surgical extirpation or x-ray therapy of the ovaries; in men, with a reduced libido. In addition, obesity often develops in pregnant and lactating females. A certain contributing role is also played by a decrease in thyroid function associated with a reduction in the basal metabolic rate.

Among the endocrine glands playing an important role in the development of obesity the pancreas ranks first. The increased excitation of the islets of Langerhans in response to alimentary stimuli is accompanied by a profuse secretion of insulin which converts excess sugar into glycogen. Other factors contributing to obesity are congenital predisposition and metabolic disorders: gout, atherosclerosis.

Certain deviations of the body weight from normal may be accounted for by specific features of the body build. As a rule, asthenic persons have a lower weight than hypersthenic ones. The development of the muscular and skeletal systems is also important. Usually, the weight of thick-set muscular persons is above normal.

Clinical picture. There is a reduction in working capacity, languor, dizziness, breathlessness, a hunger sensation, trembling of the extremities, sweats and distention of the intestine (Fig. 77).

The principal sign of obesity is a gain in weight. The question whether or not an excess in weight is present must be decided by comparison of the latter with the patient's height. There are a number of formulas for weight calculation: Broca's formula in which the height in centimetres minus 100 is equal to normal weight in kilograms; for example, for a person measuring 165 cm in height the normal weight is 65 kg; Noorden's formula in which the normal weight is equivalent to a person's height in centimetres multiplied by 420-480 and divided by 1,000, for example, the height $165 \times \times 420 : 1{,}000 = 69.3$ kg.

Obesity may vary in degree, the division into three degrees being the most convenient. In first-degree obesity the actual weight exceeds normal by 15-30 per cent, in second degree, by 30-50 per cent, and in third degree, by over 50 per cent.

Uniform distribution of fat is observed rarely. Most commonly excess fat is deposited in the chest, abdomen, back, lumbar region, nape of the neck, buttocks, thighs and in rare cases about the forearm and ankles. A patient with severe obesity is disfigured, the skin hanging down in thick folds. The skin is of a pale colour, normal or hyperaemic, with increased sweats and the development of eczema, pyodermia, and furunculosis. The cardiovascular system is most commonly affected because of a fatty infiltration of the vascular wall and the intermuscular tissue of the heart, deposition of fat in the pericardium, and displacement of the heart by a highly placed diaphragm. The cardiac sounds are muffled, and there is frequent bradycardia, myocardial dysrtophy followed by myodegeneration and circulatory failure. As a result, the patient suffers from breathlessness, palpitation, oedema, enlargement of the liver and congestive symptoms in the lungs. There are atheromatous changes in the vessels, attacks of stenocardia, myocardial infarctions, cerebral haemorrhages, etc.

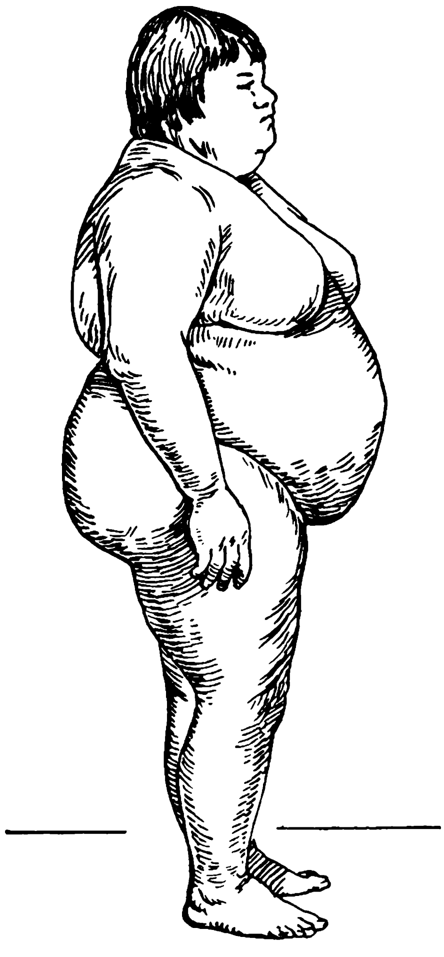


Fig. 77. Obesity

There is also a disturbance in respiratory function, a reduction in the vital and reserve respiratory capacity, with a likelihood of laryngitis, tracheitis, bronchitis, pulmonary emphysema and pneumosclerosis followed by the development of cardiopulmonary insufficiency.

The gastrointestinal symptoms are an increased appetite, gastric juice hyperacidity and salivation. There is dental caries, atrophy of the gums, frequent gingivitis and pyorrhoea. The motor function of the intestine is impaired (constipation) due to infiltration of the parietal muscle fibres with fat. There is distention of the intestine, stasis in the abdominal veins and the development of haemorrhoids. Other symptoms are hypoglycaemia and increased formation of insulin and glycogen. The liver is infiltrated with fat. In the bile ducts inflammatory changes often occur (cholecystitis and angiocholitis) and calculi form possibly due to an increased cholesterol level in the blood. Not infrequently nephrolithiasis as well as pyelitis, cystitis and urethritis develop. The joints, especially the larger ones, are also involved as a result of an increased load sustained by them as well as due to a disturbance in salt metabolism and blood and lymph circulation. In neglected cases of obesity changes are found in the spinal column and skeleton (lumbar lordosis, thoracic kyphosis, etc.).

Biochemical examination of the blood in patients with obesity shows a moderate increase in the cholesterol, calcium and uric acid levels, and a diminution in potassium.

Changes in the central nervous system are manifested by deterioration of memory, absentmindedness, easy fatigability. The peripheral nervous system is also involved while neuralgia, neuritis, radiculitis are not infrequent The latter may be secondary to spondylosis and spondyloarthrosis.

The diagnosis of obesity as an independent disease is made by excluding diseases in which obesity is only one of the symptoms. Obesity due to encephalitis, cerebral haemorrhage or traumatic lesion of the central nervous system is identified on the basis of symptoms characteristic of these diseases. In obesity caused by a disturbance in genital function (adiposogenital form) fat is deposited about the pelvis, thighs, abdomen. With an anterior pituitary tumour (Itsenko-Cushing's syndrome) fat is deposited in the abdomen, chest, nape, neck and back. There is a characteristic facies due to deposition of fat, the skin being of a purple colour. Striae of a violet colour caused by atrophy are seen on the skin of the abdomen, buttocks, thighs, neck and mammary glands. There is hypertension, increased hairiness, a metabolic disorder to the degree of diabetes mellitus. In hypothyrosis fat is uniformly distributed over the torso, the patient is somnolent, with a reduced psychic reaction. An important diagnostic sign is a lowered basal metabolic rate and a reduction in the iodine-131 uptake by the thyroid gland.

Prophylaxis and treatment. Prophylactic measures are particularly important in families liable to obesity due to congenital predisposition. A rational dietary regimen, physical culture, sports, cool showers and sponging-down procedures may prevent obesity. Persons prone to obesity must seek employment involving physical activity.

Before treatment is started it is necessary to determine the patient's weight and required caloric intake. The latter is calculated on the basis of the normal rather than actual weight. At full rest a healthy person requires 30 Cal per kg of body weight; in light work, 40 Cal, and in hard physical work, 50-60 Cal. With a weight excess of more than 10 per cent the caloric intake is restricted approximately as much as the actual weight is in excess of normal but not more than 50 per cent. Dietotherapy is carried out until the patient's weight is reduced to what is considered normal. In prescribing a diet for the obese protein products are given priority since the latter produce a specific dynamic effect. Fats and carbohydrates are restricted to a minimum because they are easily assimilated and converted into fat. The diet for an obese person must consist of rye bread, vegetables (except potatoes), lean meat, fish, poultry, lean cheese, fruit (except grapes), berries. The amount of proteins must be 110-120 g; fats, 30-35 g; carbohydrates, 200 g; the total caloricity of food should be equal to 1,600-1,800.

Thyroidin is recommended as a means to increase the basal metabolic rate. It is indicated for persons without symptoms of a circulatory disturbance, and is contraindicated for angina pectoris, myocardial infarction and liability to tachycardia. In obesity running its course with a decrease in genital function women are treated with folliculin (formula No. 168), estradiol dipropionate (oestradioli dipropionici, 0.1 per cent) in a dose of 1 ml administered intramuscularly twice weekly, and men, with a male sex hormone—testosterone propionate, methyl testosterone (formula No. 172).

The patient is enjoined to engage in vigorous physical activity improving circulation of the blood and lymph, take steam and light (sun) baths if the cardiovascular function is normal. In obesity concurrent with a cardiovascular failure due to water retention diuretics are used. These are hypothiaside in combination with potassium chloride, novurit administered intramuscularly (0.5-1 ml) twice weekly, phonurit in a dose of one 0.25 g tablet every other day.

Treatment at a health resort is also helpful.

GOUT (ARTHRITIS URICA)

Gout is a chronic disease characterized by an increased concentration of uric acid in the blood and tissues and periodic atacks of acute pains in the joints. It is usually encountered in the fourth and fifth decades of life, mainly in males.

Aetiology and pathogenesis. A certain contributing role in the development of gout is played by eating habits, in particular by overindulgence in food rich in purine bases (meat, fish) and fats interfering with the excretion of salts of uric acid (urates) with urine, and in alcoholic beverages (beer, wine, vodka). Hereditary predisposition to gout can hardly be denied.

In the pathogenesis of gout a disorder in nuclein metabolism plays a role. Nucleoproteins are an important part of the cell nucleus and in the intestine they split up into protein and nucleids. From nucleids is formed nucleic acid, and from the latter, purine bases, uric acid in particular.

Uric acid is formed in part by disintegration of the cell nuclear substance.

Gout is due, above all, to uric acid retention and accumulation in the body. Normally, a considerable part of uric acid is eliminated with urine and only an insignificant part with bile. Upon ingestion of food rich in purine bases the amount of uric acid is considerably increased, reaching a level of 2 g in 24 hours instead of the normal 0.3-0.6 g. The uric acid concentration in the blood normally ranges from 2 to 4 mg/100 ml, whereas in gout it is in excess of 5-10 mg/100 ml.

Some authors believe that the major cause of gout is an increased disintegration of nuclear substance. Others assert that the development of gout is associated with a disturbance in the uric acid elimination by the kidneys. In leukaemia, however, disintegration of nucleoproteins, though proceeding at a tremendous rate, is not accompanied by gout, neither is it encountered with a kidney disease. A contributing factor is probably a disturbance in solubility of uric acid salts and their precipitation caused by inflammatory processes in the tissues and joints. The mechanism of acute attacks of gout is also uncertain. Some authors assert that the attacks of pain, rather than being caused by deposition of uric acid salts (urates) in the tissues and joints, are due to the action of toxic substances producing an aseptic inflammation. The frequent concurrence of gout with other allergic diseases—bronchial asthma, urticaria—and the presence of eosinophilia during an attack of gout suggest the conclusion of an allergic origin of gout.

The central nervous system undoubtedly plays a role in the origination of attacks of gout. The latter's onset upon a decrease in the atmospheric pressure is possibly due to the effect of changes occurring in the nervous system.

Pathologic anatomy. There are depositions of sodium urate in the tissues and articular cartilages lying close to the surface of joints, the cartilages being destroyed and urates sometimes penetrating the underlying bones. Urates are deposited in the synovial membrane, joint capsules, tendon sheaths and cause their deformation. Deposition of urates in the soft tissues produces a reactive inflammatory process with connective tissue formation. In the periarticular tissues, cartilages of the ear, less frequently of the nose, urate depositions (tophi) enclosed in a capsule are formed.

Clinical picture. An attack usually begins at night or at dawn with an intense pain in the metatarsophalangeal joint of the big toe, less frequently in the talocrural, metatarsophalangeal, elbow and radiocarpal joints of the hands. There is a simultaneous rise in temperature to 40°C with a chill. An attack may be provoked by mental and physical overexertion, a change in the atmospheric pressure as well as ingestion of food rich in purine bases or abuse of alcohol. Pains are localized to one or several joints. Within a few hours the joint becomes swollen and the skin over it turns purple-red. The joint is extremely tender and sensitive to palpation, tense, lustrous and hot. The blood exhibits an elevated ESR, neutrophilic leucocytosis and eosinophilia. An attack is sometimes accompanied by a loss of appetite, nausea, vomiting, eructation, heartburn, headaches and dizziness. It may last for 5 to 6 days at half-an-hour to one hour and longer intervals, then pains gradually abate, the swelling subsides, the skin regains its normal colour, and the amount of uric acid, which during an attack is excreted in quantity, is considerably diminished. Sometimes a renal form of gout is encountered when uric acid salts (urates) are deposited in the kidneys, particularly in the medullary substance, with the development of inflammation about these depositions. Not infrequently the skin over a tophus is atrophied with an eruption of a whitish mass containing sodium urate.

In time, frequent recurrence of attacks causes considerable changes in the articular ends of bones and in the joints proper. Gouty nodes form in the joint capsules, the joints become disfigured, while in severe cases contractures, dislocations and subluxations occur. The patient becomes disabled.

The x-ray usually shows distinctly outlined structureless round or semicircular foci in the epiphyses of bones in direct proximity to the joint surface, bone defects resulting from resolution of bone tissue at the site of deposition of uric acid salts. The diagnosis of obliterated or atypical forms is aided by a test load with overingestion of food rich in uric acid (thymus, liver, etc.), when a delayed excretion of uric acid is seen.

Gout is often concurrent with hypertensive vascular disease, atherosclerosis and obesity. Gouty persons are predisposed to respiratory diseases (bronchitis, bronchial asthma). They frequently have pyelitis, pyelocystitis, nephrolithiasis and cholelithiasis, myalgia, neuralgia, an enlarged and tender liver.

The diagnosis of gout should be made upon an increase in uric acid concentration in the blood after maintaining the patient on a purine-free diet for three days, in the presence of a characteristic x-ray picture and tophi. Care should be taken lest the latter be mistaken for Heberden's nodes—symmetrically arranged thickenings of the terminal phalanges of the fingers due to an endocrinic disorder (climacteric).

Prognosis depends on the attendant atherosclerosis of the coronary, cerebral and renal vessels, and articular lesions. The patient retains his working capacity in full if no serious changes are present in the joints and vascular system.

Prophylaxis and treatment. A patient with a metabolic disorder should avoid overeating, restrict the intake of foods rich in purine bases, saccharine substances (sweets), fats, and abstain from alcohol. Sports, physical culture and light physical work are recommended. In an acute attack of gout complete bed rest is mandatory. Complete or partial fasting during 1-3 days, milk dishes, abundant drink and thin jellies are prescribed. Drug therapy consists of administrations of tincture of autumn crocus (Tincturae Colchicini) in a dose of 10-15 drops to be taken every 2-3 hours until an abatement of pains, atophan (cinchophen) in a dose of 0.5 g given 3-4 times daily during a period of 2-3 days. Atophan treatment may be resumed after a 7-to-10 days' interval. One should bear in mind the latter's possible toxic effect, particularly on the liver. Atophan treatment is combined with an abundant intake of liquid—3 or 4 glassfuls of alkaline water daily. The latter prevents precipitation of uric acid salts, increases uric acid excretion by the liver and mobilizes uric acid salts accumulated in tissues for their excretion with urine. Sodium salicylate in a dose of 6 g in 24 hours, butadione in a dose of 0.15 g 2-3 times daily, adrenocorticotropic hormone, prednisolone, vitamin B_1 and urodan may also be prescribed. Highly efficacious is treatment with ethamide in doses of 0.3-0.5 g in 24 hours, the total dose amounting to 10-15 g for a treatment course. During ethamide treatment administrations of aspirin and salicylates are forbidden because they reduce the former's effect. Ethamide fails to mitigate pains and reduce inflammation in the joints. Apparently, just as adrenocorticotropic hormone, salicylates and atophan, it hinders an increased absorption of uric acid in the renal tubules.

In periods of remission treatment consists in restrictions on foods containing purine bases—animal proteins, meat, fish, particularly thymus, liver, kidneys, brains, meat broths and fish soup as well as certain vegetables, such as sorrel, spinach, radish, peas, beans and lentils. Treatment at a health resort aids recovery.

Alimentary Disorders. Vitamin Dificiency

ALIMENTARY DYSTROPHY

Alimentary dystrophy was formerly called nutritional dropsy because of the oedema caused by undernourishment. It was also known as protein-deficient oedema since an association had been established between hunger oedema and protein deficiency in the body. The term "alimentary dystrophy" was coined by Soviet authors during World War II. The well-known Russian pathologist V. Pashutin was the first to describe the pathology of hunger oedema, while M. Manassein, who in his time studied under S. Botkin, described the clinical manifestations of this disease.

Aetiology and pathogenesis. Alimentary dystrophy results from malnutrition, an inadequate supply of proteins, fats and carbohydrates to the body. The leading role in the genesis of this disease, however, is played by a deficiency of nutritious proteins in food.

Highly important factors are also the condition of the central nervous system and psychic traumas, as well as physical overexertion and chilling which increase energy expenditure. This disease is usually encountered in males. Persons with obesity starving for a long time are safeguarded against alimentary dystrophy by their large reserves of fat. An undernourished person consumes first of all his reserves of fat, then the glycogen of the liver and muscles, and later proteins of the muscles and parenchymatous organs. A diminution of protein reserves causes atrophy of the organs and tissues. Atrophy of the striated and smooth muscles causes muscular and intestinal weakness. Atrophy of the sebaceous, sudoriferous, and salivary glands entails dryness of the skin. The intestinal and gastric secretion is depressed. Atrophy of the liver and spleen reduces body resistance to infectious diseases.

Alimentary dystrophy causes endocrinic disorders and vitamin deficiency due to an inadequate supply of vitamins with food as well as their poor assimilation in the body. The basal metabolic rate is reduced, which indicates adaptation of the body to undernourishment.

Pathologic anatomy. Atrophy involves the fatty subcutaneous tissue, skeletal muscles, the liver, the spleen, and the heart muscle. Marked changes occur in the organs with a diminution of their weight. The mucous membrane of the gastrointestinal tract is severely atrophied. Atrophy affects the adrenal cortex, the mammary gland, the genital glands, and partly the thyroid. Enteritis and tuberculosis of the lungs and lymph nodes are often discovered at autopsy.

Clinical picture. The early signs of alimentary dystrophy are an increased appetite, lassitude, pains and a feeling of heaviness in the leg muscles, frequent micturition, and nycturia. There is urinary urgency which demands immediate satisfaction to avoid the danger of incontinence. Impotence develops in males, and amenorrhoea in females.

Upon inspection the patient's aged appearance and severe pallor of the skin strike the eye, the skin being dry and coming off in scales. The patient lies motionless for hours, huddled up under a blanket pulled over his head. There is severe emaciation, a considerable wasting of the subcutaneous cellular tissue to the degree of its complete disappearance, and atrophy and weakness of the muscles (Fig. 78).

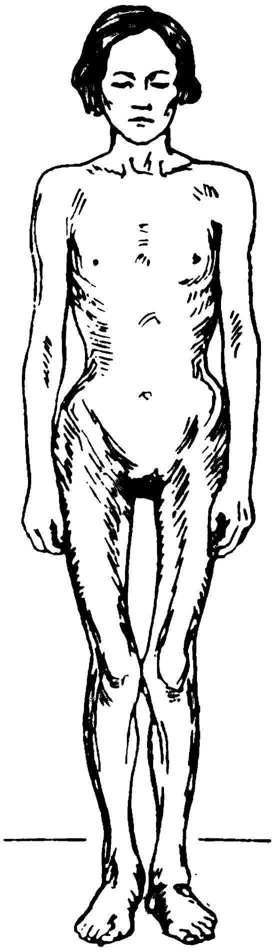


Fig. 78. Emaciation

Oedema in alimentary dystrophy may be early or late. Early oedema develops a long time before the weight loss, is unstable and rapidly subsides after the patient has been prescribed bed rest and warmth. It is concurrent with polyuria and often appears after a physical strain. Late oedema develops within 2-3 months of the onset of the disease in the presence of manifest symptoms of cachexia. It varies in degree from a pastiness to a massive oedema. It spreads throughout the body and fills the serous cavities (ascites, hydrothorax, etc.). Late oedema results from protein deficiency in the blood.

The cardiovascular symptoms are bradycardia, hypotension, dull cardiac sounds, and weak heart contractions.

Highly characteristic changes are found in the gastrointestinal tract. An increased appetite persists for a long time but anorexia is often present in the terminal stages of the disease. Gastric secretion is decreased or a total absence of hydrochloric acid is noted. At first the patient is discomforted by constipation, and later an exhausting diarrhoea develops due to a failure of gastric function and sometimes due to pellagra or concurrent enterocolitis.

Severe changes also occur in the neuropsychic sphere.

The patient is in a torpor, answers questions slowly and with a delay; he is apathetic and indifferent to himself and his surroundings, and fairly often has an acute psychosis and hallucinations.

The basal metabolic rate is lowered in proportion to the severity of alimentary dystrophy. The blood sugar level is reduced to 50-60 mg/100 ml. The protein content is also decreased to 3-4 g/100 ml instead of the normal 6-8 g/100 ml. The amount of calcium and cholesterol in the blood is also diminished. A slight hypochromic anaemia and leucocytosis are encountered sometimes.

Course and outcome. The dry or nonoedematous form of the disease runs a more severe course and is often fatal. The course of the oedematous form is more favourable if rational therapy has been given in due time.

Alimentary dystrophy is not infrequently complicated by bronchitis, pneumonia, infectious enterocolitis, commonly of dysenteric origin, and pulmonary tuberculosis. In the majority of cases, the above complications run their course without a rise in temperature and changes in the blood. Even a slight fever, however, and elevation of the ESR are highly suspicious.

Alimentary dystrophy should be differentiated from certain diseases producing similar outward signs. Acute nephritis differs from alimentary dystrophy by the presence of protein, erythrocytes, and casts in the urine. In amyloid-lipoid nephrosis, also running its course with a massive oedema, albuminuria and hypercholesterolaemia occur. Cardiac oedema is distinguished by symptoms of cardiac failure.

The dry form of alimentary dystrophy must be differentiated from cachexia due to a malignant neoplasm, intoxication, and endocrinic disorders.

Treatment. The patient is given complete rest and warmth (heat applications). In mild cases a good effect may be produced by prescribing a mixed diet including nutritious proteins (meat, milk, cottage cheese, eggs, lentils, peas, kidney beans), carbohydrates and fats (not more than 50 g), with a reduced intake of common salt. Food should be given in small portions 5-7 times a day. To increase protein intake 25 g of haematogen (defibrinated blood preparation. *Ed.*) should be administered. The intake of common salt is restricted only in oedematous forms. Patients with far-advanced forms of alimentary dystrophy are maintained on a bland, easily assimilated food stimulating the neurovascular system: coffee, sweetened tea, broth; later the diet is gradually supplemented with mucilaginous soups, thin jelly, jelly, and rusks. In diarrhoea mechanically and chemically irritating food must be excluded from the diet. The patient's menu should contain foods rich in calcium salts, i.e., milk and eggs. Drug therapy consists of intravenous injections of 20-40 ml of a 40 per cent glucose solution and 10 ml of a 5 per cent calcium chloride solution, and administration of vitamin C in a dose of at least 500 mg daily, vitamins A and those of the B complex. Blood transfusions in doses of 80-100 ml are recommended only for patients in a moderately severe condition. In anaemia iron and vitamin B_{12} are prescribed; in pellagra, nicotinic acid (formula No. 155); in a circulatory disturbance, caffeine and adrenalin subcutaneously.

AVITAMINOSIS AND HYPOVITAMINOSIS

Avitaminosis and hypovitaminosis are diseases due to a total or partial deficiency of vitamins in the body. They are caused by an inadequate vitamin supply with food, malabsorption in the gastrointestinal tract, and diseases of the liver since the latter contributes to the production of a number of vitamins. Avitaminosis and hypovitaminosis also result from great physical overstrain, overheating, chilling; they may occur in pregnancy and lactation, as well as in consequence of intensified vitamin destruction in the body in various infectious diseases. Vitamin deficiency causes a disturbance in vital functions: cellular respiration, carbohydrate and mineral metabolism, production of hormones, capillary permeability, etc. Vitamin deficiency is encountered in a manifest or concealed form: the latter produces no striking clinical picture but is very important practically.

Avitaminosis A. This disease results from partial or total absence of vitamin A from the food as well as in cases of a disturbed convertion of carotene (provitamin A) into vitamin A. Vitamin A deficiency manifests itself by a variety of changes in the organ of vision, skin, upper air passages, urinary passages, and gastrointestinal tract. The skin and mucous membranes, particularly those of the eyes and upper air passages, become cornified, dry, and peel off in scales.

One of the earliest symptoms of vitamin A deficiency is night blindness (nyctalopia) which results from a disordered formation of visual purple (rhodopsin). At dusk there is a considerable loss of visual acuity and at night the patient is unable to discern objects. In the daytime there is a reduction in photoreception and the colour sense, particularly for yellow and blue. On the conjunctiva there are whitish or yellow spots failing to be moistened with tear secretion. The dryness extends to the entire conjunctiva and xerophthalmia ensues. In severe cases ulcers producing keratomalacia form in the cornea. The prognosis in night blindness is favourable only in the initial stage; in far-advanced cases, i.e., in keratomalacia, an irreversible loss of vision is likely.

Severe changes occur in the skin and the sudoriferous and sebaceous glands. The skin is intensely pigmented and peels off in scales, while the follicles are cornified. Laryngitis and hoarseness, tracheitis, bronchitis, and stomatitis are encountered. The gastrointestinal symptoms are achylia and diarrhoea. Not infrequently cystitis, urethritis, and renal calculi occur.

Treatment should above all ensure an adequate supply of vitamin A or carotene to the body. The patient is maintained on a diet containing animal fats (except lard), butter, cod-liver oil, fat grades of meat, liver, fish, eggs, milk and dairy products. Food rich in carotene (red carrots, tomatoes, black currents, green peas, apricots, spinach) is also prescribed. Nyctalopia is cured within 2-5 weeks of treatment with cod-liver oil given in a dose of 3 tablespoonfuls daily. In the treatment of xerophthalmia and keratomalacia cod-liver oil is administered for a long period. For therapeutic as well as prophylactic purposes 0.5-1 mg of vitamin A or 1-2 mg of carotene (formula No. 165) is added to food. The daily vitamin A requirement of a healthy person is equal to 3 mg.

Avitaminosis B_1. Avitaminosis or hypovitaminosis B_1 results from deficiency of vitamin B_1 in the food or its increased disintegration in the body during infectious and inflammatory diseases, from a prevailing carbohydrate diet, or insufficiency of fats in the food. Avitaminosis manifests itself by a disease known as beri-beri running its course in an oedematous, cardiac or polyneuritic form. The oedematous form produces widespread oedema in the subcutaneous cellular tissue and visceral cavities. The cardiac form may cause a circulatory disorder, with expanded heart boundaries, muffled cardiac sounds, and sometimes with a disturbance in conduction.

The polyneuritic form is manifested by disturbance in sensibility, weakness and pains in the extremities, unsteady gait, and muscular atrophy. Paralysis develops in severe cases. In all forms of hypo- or avitaminosis B_1 there is constipation and meteorism due to intestinal atony, a loss of appetite, and pain in the extremities.

Vitamin B_1 deficiency is prevented by prescribing a diet rich in this vitamin: rye and coarse white bread, various gruels, pulses, liver, brewer's yeast, tomatoes, oranges, lemons, grapes, and fresh meat. Treatment also includes intramuscular injections of a 1-5 per cent vitamin B_1 solution in a dose of 1 ml (formula No. 156). Vitamin may also be prescribed in dragée and tablet form. The daily dose of vitamin B_1 for a healthy person is 3 mg.

PELLAGRA

Pellagra (It. *pella*—skin, *agra*—rough) occurs as the result of a dietary deficiency of nicotinic acid, the pellagra-preventing P.-P. factor which is included in the vitamin B complex. Pellagra is also encountered in a number of infectious diseases: dysentery, tuberculosis of the intestines, protracted forms of enteric fever, sepsis, etc.

In pellagra protein and carbohydrate metabolism is disturbed and the amount of iron in the body is reduced.

The disease is encountered in a severe or concealed form. The symptoms of the former are as follows: (a) skin lesions—dermatitis; (b) disorders in the alimentary tract, in particular diarrhoea; (c) derangements in the neuropsychic sphere—dementia (imbecility). The skin, mainly its exposed areas, i.e., on the face, neck, and hands, is covered with red spots (erythema), resembling sunburns, which in time turn brown. Later the skin begins to peel off in scales and becomes covered with vesicles filled with serous or purulent contents. Lesions of the skin occur symmetrically over the extremities and neck. In these parts the skin becomes dark in colour, the legs seeming to be dressed in dark stockings, the hands in gloves, with what looks like a collar about the neck. Hence the terms pellagrous stockings, gloves, collars.

There is a loss of appetite, burning of the tongue, and salivation. The tongue is smooth and lustrous as though lacquered as a result of atrophy of the papillae; it is bright-red in colour and is covered with a brown coat and fissures. The gastrointestinal symptoms are achylia and diarrhoea with malodorous stools. In severe cases the above symptoms are joined by disorders in the nervous system, with a deterioration of memory, a derangement in all types of sensibility, the patient becoming slow-witted, reticent, and indifferent to his surroundings. Full-blown psychosis—hallucinations and depression—are also encountered sometimes.

An acute form of pellagra may prove fatal. The disease usually runs a chronic course with periodic relapses and remissions.

The most efficacious therapeutic measure is a timely prescription of a diet containing vitamins of the B complex (meat, liver, fish, eggs, cottage cheese, green peas, beans, tomatoes, lettuce). The food is supplemented with 50-100 g of dry brewer's yeast which is taken in equal portions several times a day. Drug therapy consists of administrations of vitamin PP in the form of nicotinic acid in a dose of 0.05 g three times daily or intramuscular injections of a 1 per cent nicotinic acid solution in a dose of 3-5 ml during a period of 10-15 days. For gastric achylia hydrochloric acid is prescribed.

SCURVY (SCORBUTUS)

Scurvy occurs as the result of a prolonged dietary deficiency of vitamin C—ascorbic acid. It may also be caused, however, by infectious diseases in which the vitamin C requirements of the body are increased, and by gastrointestinal diseases in which the vitamin is intensively destroyed or fails to be assimilated and is excreted in the faeces.

The factor underlying all clinical manifestations of scurvy is an increased capillary permeability. Scurvy occurs in a mild, moderately severe, or severe form. In a mild form the patient complains of general malaise, debility, easy fatigability, and dragging pains in the gastrocnemius muscles. The skin is rough and peels off in scales, the gums are spongy and bleeding, and punctate haemorrhages appear below the site of compression of the arm with a tourniquet. Upon transition of mild scurvy into a moderately severe form the abovementioned signs are joined by multiple punctate haemorrhages about the hair follicles, seen as petechial rashes, and by considerable extravasation of blood in the cellular tissue and about the joints in the form of large ecchymoses. Extravasations into the muscles, into those of the femur in particular, are frequent. The gums are covered with an ashen-grey coat and are sometimes ulcerated. The teeth get loose and fall out. There is a foetid odour from the mouth, and the cervical and submandibular lymph nodes are enlarged.

Severe forms of scurvy are attended with more extensive haemorrhages into the depth of the muscles, in the subcutaneous cellular tissue, and in the serous cavities (pleural, abdominal, etc.). There is anorexia, nausea, and diarrhoea; the stools not infrequently become bloodstained. In severe cases hypochromic anaemia and severe emaciation develop.

If the diagnosis has been established in good time scurvy may be cured within 2-3 weeks although increased capillary permeability persists for months. Treatment consists in maintaining the patient on a diet rich in vitamin C. The following food products are recommended: cabbage and sauerkraut, spring onions, lettuce, young potatoes, black currants, garlic, lemons, fruit, fruit juices, etc. In addition, an infusion of dry sweetbrier, green pine needles, and birch and lime-tree leaves is prescribed. A 5 per cent ascorbic acid solution in a dose of 1 ml (formula No. 157) is administered intravenously during the first ten days; later, after the body has been saturated with vitamin C its daily dose is lessened to 50-100 mg (tablets, dragée). Gargling of the mouth and painting of the gums with disinfectant and astringent solutions—manganese peroxide, rivanol and alum—are helpful also.

Diseases of the Organs of Movement

AN OUTLINE OF ANATOMY AND PHYSIOLOGY

Of the organs of movement the joints are the most frequently affected since circulation of the blood is developed poorly in them. In addition, they are more liable to injury. The joints are also exposed to the influence of toxicoallergic and infectious factors which interfere with their function. A disease of a joint may also result from a metabolic disorder when the end products of metabolism subject to elimination from the body are deposited in tissues about the joints, possibly because of a poor circulation of the blood within the latter.

Every joint consists of (a) epiphyses (ends of articulated bones) covered with cartilage; (b) joint cavity; (c) joint capsule.

The articular cartilage consists of hyaline and has an elastic consistency and a smooth inner surface. It receives its nutrition from the synovial fluid because it has neither blood vessels nor nerves. The joint cavity contains synovial fluid which, in addition to supplying the cartilage with nutriment, facilitates movement of the joints. The joint capsule consists of an external fibrous capsule and an inner synovial membrane. The fibrous capsule is connected with the periarticular tissue, while the synovial membrane which has a rich network of lymph and blood vessels, secretes synovial fluid. Of the variety of existing classifications of articular diseases, the best known is the one in which all principal forms of diseases of the joints are divided into 5 groups.

1. **Infectious arthritis.** This group includes: (a) rheumatic polyarthritis; (b) rheumatoid, or nonspecific, polyarthritis due to a nonspecific focal infection in the teeth, maxillary sinus, tonsils, gallbladder, urogenital system, etc.; (c) infectious specific polyarthritis of a definite aetiology (due to tuberculosis, gonorrhoea, brucellosis, dysentery, syphilis, influenza, fevers, etc.).

2. **Dystrophic (noninfectious) arthritis:** occupational arthritis, deforming arthritis, spondylosis, gout, neurotrophic arthritides (tabetic, those seen in paralysis, lesions of the spinal cord, injuries

to the peripheral nerves); dystrophic arthritis associated with endocrine disturbances (thyrotoxic, ovariotoxic, those seen in acromegaly, etc.).

3. **Traumatic arthritis.**
4. **Arthritis associated with other diseases.**
5. **Rare forms of articular lesion** (serum- and drug-induced forms).

In respect of their course arthritides are subdivided into acute, subacute, and chronic. For the degree of disability the following forms of functional failure are known: mild (I), moderately severe (II), and severe (III).

INFECTIOUS NONSPECIFIC, OR RHEUMATOID, POLYARTHRITIS

Rheumatoid polyarthritis is an independent disease encountered fairly frequently, mainly at a mature and elderly age.

Aetiology and pathogenesis. The aetiology of this disease is uncertain. It is commonly believed that rheumatoid polyarthritis is caused by a focal streptococcal infection—tonsillitis, sinusitis, otitis, appendicitis, cholecystitis, pyelitis, adnexitis, prostatitis, etc. The predisposing factors are physical or mental overstrain, intoxication, and common cold. A role is apparently also played by the state of the endocrine system, in particular the pituitary body and adrenal cortex. Thus, the origin of rheumatoid arthritis is of a complex character associated with infectious-allergic and neuroendocrinic factors.

Rheumatoid polyarthritis causes a systemic affection of the connective tissue, its fibrinoid swelling and necrosis, as a result of which collagen fibres disappear and are replaced by an amorphous substance. At the same time there is an increase in capillary permeability, the plasma protein passing from the vessels into the surrounding tissue and causing nonspecific sensitization of the body, which contributes to the formation of autoantibodies.

At present two stages are distinguished in the course of rheumatoid polyarthritis: (a) the exudative form which corresponds to the onset of inflammation of the synovial membrane and the appearance of a thick exudate permeating the periarticular tissue, and (b) the fibrinous form which corresponds to the onset of granulation tissue formation, followed by the development of scar tissue and limitation of mobility of the joint. The disease process sometimes produces only an inflammation of the synovial membrane and is reversible. Most commonly, however, the inflammation extends to the ligaments, periarticular structures, cartilage, and bone with destruction of cartilage, development of ankylosis, contraction of ligaments and tendons, and sometimes dislocations and subluxations.

Clinical picture. The disease sets in with acute pains in the joints and fever. The joints of the extremities are usually symmetrically involved, and often the joints of the jaws and spine are also affected. An inflammatory and sometimes haemorrhagic exudate accumulates in the joints. Pains may become so intense as to be relieved with narcotics only. The capsule and periarticular tissues are thickened, while ulceration of cartilages and their replacement with fibrous tissue are noted fairly often. The joints are swollen and deformed, the skin over them is taut, and the muscles adjoining the diseased joints are atrophied. The disease often runs a chronic course with frequent relapses. The periarticular thickening limits the mobility of the joint or causes ankylosis—complete immobility. Usually the wrists, elbows, hips and knees are fixed in a flexed position, and the shoulder joint, in an adducted position. Ankyloses and contractures of the joints ultimately lead to atrophy of the skeletal muscles.

An affection of the spine causes ossification of the structures interconnecting the vertebrae, which results in the development of the so-called bamboo spine, ankylosing spondylitis, otherwise known as Bechterew-Marie disease. This produces kyphosis of the thoracic part of the spinal column and complete rigidity of the neck. In nonspecific (rheumatoid) polyarthritis in addition to lesions of the joints there may be a number of other symptoms: enlargement of the lymph nodes, liver and spleen, leucopenia, anaemia, and an elevated ESR (Felty's syndrome), and involvement of the kidneys and the thyroid gland. Sometimes an acute form of rheumatoid polyarthritis concomitant with conjunctivitis and urethritis (Reiter's syndrome) is encountered.

Course. The course of rheumatoid polyarthritis may be acute, subacute, or chronic. With a chronic course there are periodic relapses due to infection and chilling.

Differential diagnosis between rheumatic and rheumatoid polyarthritis presents certain difficulties. However, they have substantial distinctions which may be used as a guide to a correct diagnosis. Rheumatic polyarthritis commonly occurs at a young age and mainly affects the larger joints. As a rule, there are no deformities or contractures. Limitation of mobility, if it is present at all, is seen only in an acute period. Highly characteristic symptoms of rheumatic polyarthritis are wandering pains and swelling of the joints, trophic changes in the muscles and skin being unmarked and rheumatic endocarditis and a heart defect being present.

Rheumatoid polyarthritis is mainly encountered at a mature and elderly age. The disease at first affects the small and then larger joints, and deformities, ankylosis, and atrophic changes in the muscles and skin occur. The pains in the joints are not wandering, while cardiac involvement is absent or very slight. Both rheumatic and rheumatoid polyarthritis is attended with a high-grade fever and an elevated ESR. Salicylates are more efficacious for rheumatic polyarthritis.

Prognosis is favourable in respect of survival and varies with the functional condition of the joints in respect of working capacity. Three degrees of functional disablement of the joints are distinguished. With the first degree there is only an insignificant limitation of motion in the joints without a marked reduction in working capacity. With the second degree movements in the joints are so restricted that the patient is unable to work and care for his needs. The third degree is characterized by complete immobility of the joints, the patient being quite helpless.

Prophylaxis and treatment. Prophylaxis is difficult because of the uncertain aetiology of the disease. Nevertheless, a systematic treatment of chronic foci of infection—tonsillitis, sinusitis, septic foci of any localization—is necessary. In addition, general prophylactic measures must be taken against diseases of the joints, described in the chapter on rheumatic fever (favourable living, housing and climatic conditions, hardening of the body, moderation in physical and mental work).

Treatment. The primary measure is complete bed rest. Sulpha drugs and antibiotics (penicillin, streptomycin, tetracycline, etc.) are prescribed, since the disease is essentially due to infection. In addition, antiallergic (desensitizing) agents—salicylates, pyramidon, butadione—are used (see in the section dealing with the treatment of rheumatic fever). During the past few years good results have been obtained with cortisone, prednisone, prednisolone, and ACTH. Cortisone is prescribed in a dose of 100-200 mg daily (50 mg 3-4 times daily) for a period of 2-3 weeks. As the patient's condition becomes better, the daily dose may be reduced to 50 and 25 mg. ACTH is administered intramuscularly in a dose of 20 U twice daily. During ACTH and steroid hormone therapy there is retention of water in the body; therefore, the patient is maintained on a salt-free diet and is sometimes given diuretics (novurit, phonurit, hypothiaside). To enhance the effect, a combined therapy with salicylates, butadione, pyramidon, rheopyrine and corticosteroid hormones is indicated. In recent time success has been gained in the application of resochin (antimalarial drug), particularly in the early period of the disease, in a dose of 0.25 g twice daily given one hour after a meal. Resochin produces no untoward effects and can be administered for a long time (up to one year). Rheumatoid polyarthritis is treated by intramuscular injections of crisanol (5-10 per cent oil suspension of calcium aurothiopropanolsulphonate and calcium gluconate). In Felty's syndrome splenectomy is recommended. In chronic forms of rheumatoid polyarthritis physiotherapy and balneotherapy, iono-galvanization with potassium iodide, salicylates or histamine, massage, therapeutic gymnastics, and ozocerite and paraffin applications are prescribed. Sulphur and radioactive-water baths are very helpful. In chronic polyarthritis treatment at a health resort is beneficial.

INFECTIOUS ARTHRITIS OF PROVEN AETIOLOGY

In many infectious diseases acute serofibrinous inflammations of the joints occur in different periods; they may be specific or nonspecific in character. The affected joint becomes disfigured by an accumulation of exudate in its cavity and periarticular oedema. In addition, the joint is thickened as a result of proliferation of connective tissue in the capsule itself. At the same time x-rays reveal a narrowing of the joint space, distention of the joint with exudate, rarefaction of bone and its proliferation in the form of osteophytes, spurs and ossification of the ligamentous apparatus.

Infectious arthritis is consequent upon gonorrhoea, syphilis, tuberculosis, streptococcal, staphylococcal, and pneumococcal diseases, scarlet fever, dysentery, enteric fever, paratyphoid, Botkin's disease, brucellosis, etc.

The mechanism of affection of the joints is dissimilar. In some cases inflammation of the joints results from a direct penetration of the pathogenic agent into a joint (metastatic form), in other cases the disease process in a joint is conditioned by toxaemia, and in certain cases, by allergic factors.The character of affection of the joints varies with the aetiology of the disease. In septicopurulent processes there is metastatic purulent lesion of one or several joints.

Clinical picture. The manifestations of infectious arthritis are varied. A lesion of the joints occurs in different periods of an infectious disease but sometimes precedes the infection itself, as is the case in Botkin's disease. In some cases infectious arthritis manifests itself in the period of convalescence (in dysentery, lobar pneumonia, etc.) or at the peak of the disease.

The clinical picture of infectious arthritis in the majority of cases differs from that of rheumatic arthritis. The affection involves a smaller number of joints than in rheumatic polyarthritis and does not wander from joint to joint. In certain cases, the disease runs a chronic course, involving also smaller joints and ultimately causing deformities, stiffness, or ankylosis.

The treatment of infectious arthritis is mainly conservative, except that in metastatic and specific forms in which aetiologic therapy and surgery are indicated.

SEPTIC ARTHRITIS AND POLYARTHRITIS

In septic diseases metastatic lesions of one or several joints may be encountered. In such cases, the purulent exudate in a joint may harbour the causal organism of sepsis. Sometimes the articular exudate is serofibrinous in character.

Treatment is carried out according to the general rules of management of sepsis. Sulpha drugs or antibiotics are prescribed after preliminarily testing the sensitivity of the pathogenic agent to a certain antibiotic. The joints are put in a normal position and exercise of the extremities are prescribed in order to avoid ankylosis and deformities. Physiotherapy, hydro- and mud therapy are also used. In the presence of pus in a joint, which is detected by puncture, surgery and local therapy are required.

Prognosis varies with the course of the principal disease. Ankylosis is frequent, especially in purulent metastatic forms which make the patient incapacitated.

PNEUMOCOCCAL ARTHRITIS

Pneumococcal arthritis is most commonly isolated in character (monoarthritis) and is mainly encountered with lobar pneumonia, although in separate cases it may occur also in pneumococcal tonsillitis, otitis, sinusitis, etc.

Mild forms of pneumococcal arthritis heal spontaneously. Arthritis running a protracted course is cured with sulpha drugs and antibiotics. Sometimes puncture of a joint and its lavage with antibiotics are used, and in the presence of pus surgery is indicated. Upon abatement of acute symptoms light passive and active movements, physiotherapy and balneotherapy are prescribed.

Prognosis is usually favourable, but in the purulent forms and complications it is serious.

Prophylaxis consists in prevention and timely treatment of the principal disease.

GONORRHOEAL ARTHRITIS

Gonorrhoeal arthritis develops in the majority of cases within three weeks and sometimes several months of the onset of gonorrhoea. It sets in with acute pains in many joints and a rise in temperature to 38-39°C. Later the process becomes localized to one or two larger joints, usually the knee, the ankle or the wrist. The affected joint is severely swollen, there is periarticular oedema, and an intense pain on the slightest touch or movement. Exudate forms in the joint capsule. Very soon atrophy of muscles closest to the joint develops, the cartilage is sometimes destroyed, and the bones near the cartilage become loose in structure. High fever persists not longer than a week, and later the disease runs its course with a normal temperature. The disease lasts from several weeks to several months and ends in deformity and ankylosis of the joint.

In the acute period the patient should be at complete bed rest and the affected joint must be immobilized for some time. Sulpha drugs are prescribed in a daily dose of 4-6 g during a period of 5-7 days, not infrequently in combination with antibiotic injections (penicillin). Should these remedies fail or prove of little therapeutic effect a gonococcal vaccine must be administered, starting with small (25 mg) and ending with large doses (500 mg). It is recommended to give 10 injections for a course of treatment at intervals of 3-4 days. In the acute period a local application of heat to the affected joint is indicated. Analgetics—pyramidon with aspirin in equal doses of 0.25 g 3-4 times daily, and analgine in doses of 0.5 g three times daily in combination with luminal (0.05 g) are also prescribed. Quartz light irradiation (ultraviolet light) in erythema doses is indicated.

In case of transition of an acute form into a subacute process passive and active exercises of the affected joints and massage of the muscles are prescribed in order to avoid ankylosis and atrophy of the skeletal muscles. In addition, upon abatement of the initial symptoms an intensive heat thermotherapy (diathermy, Sollux) as well as mud, paraffin, ozocerite and peat applications and balneotherapy (hydrogen sulphide baths) are prescribed.

Prognosis is favourable if treatment is correct and given in due time, and serious in a purulent form which may result in ankylosis. In view of this, after abatement of acute symptoms limitation of movement of the affected joint for a long period is recommended. A chronic process may lead to ankylosis of the joint and incapacity.

Working capacity in mild cases is regained within one to several months. In chronic processes and complications working capacity is estimated from the patient's condition.

Prophylaxis consists in measures against gonorrhoea and a timely and vigorous treatment of this disease and its complications.

TUBERCULOSIS IN THE JOINTS

Tuberculous lesions of the joints result above all from a specific inflammation due to the entry of *Mycobacterium tuberculosis* into the joint from the blood flow. One example of such lesions is tuberculous coxitis or as it is also known "a white tumour of the joint". In this condition the disease process is localized to only one joint. Tuberculous dropsy affects mainly the knee. Tuberculosis in the joints develops slowly, running a chronic course, and is attended with atrophy of the muscles near the affected joint, stiffness and sometimes ankylosis of the latter. The disease is treated surgically. Tuberculosis, however, causes a number of changes which are manifestations of the general disease, possibly due to toxic and allergic factors.

In these cases there is multiple involvement of the knees, ankles, and wrists. This condition was for the first time described by Poncet; it is known as tuberculous rheumatism and is a nonspecific infectious polyarthritis. The patient is pale, has a decreased appetite and subfebrile temperature.

Treatment. The primary measure is systematic treatment of tuberculosis. Streptomycin, phthivazide, PASA and general invigorating measures (air baths, sun baths, vitamins C and D, nutritious diet with a rich content of vitamins and calcium salts) are prescribed.

After abatement of fever, massage and regulated exercises are necessary.

The *prognosis* of tuberculous polyarthritis is usually favourable. Local specific lesions of the joints not infrequently cause ankylosis which markedly reduces the patient's working capacity to the degree of complete disability.

Prophylaxis. This consists in general measures against tuberculosis.

SYPHILITIC LESIONS OF THE JOINTS

Syphilitic lesions of the joints occur in secondary and, in a more pronounced form, in tertiary syphilis. One or two joints, most commonly the knees and ankles, are involved. In some cases the clinical picture is dominated by changes in the synovial membranes; in other cases the bones, osteocartilaginous and periarticular structures are affected. A syphilitic lesion of the joints resembling for its course rheumatoid arthritis is also known. Syphilitic lesions of the joints are symmetrical in localization and cause intense pains at night. An important diagnostic sign is not only the character of affection of the joints but also a history of syphilis and a positive Wassermann's test.

In conducting specific therapy (bioquinol, salvarsan, etc.), preference is given to iodine preparations and antibiotics (penicillin, and others). In the acute period local applications of heat to the diseased joint (paraffin applications, dry air baths) are used. Massage is contraindicated in the presence of acute and subacute symptoms. Severe pains are mitigated with pyramidon, analgine, aspirin, etc. Upon abatement of acute symptoms sulphur baths, as well as physiotherapy (diathermy, etc.) are prescribed.

Prognosis is unfavourable in neglected cases with a severe course in which deformities of the joints causing considerable limitation of movements develop.

Working capacity is temporarily reduced.

Prophylaxis consists in measures against venereal diseases and in a timely and vigorous treatment of syphilis.

SCARLATINAL POLYARTHRITIS

A lesion of the joints in scarlet fever usually occurs at the end of the second or during the third week of the disease. For its clinical course scarlatinal polyarthritis resembles rheumatic polyarthritis. It is more frequently encountered in adult persons and is often complicated by endocarditis, for which reason it is considered to be rheumatic in origin. Treatment is conducted with salicylates which are highly efficacious and with steroid hormones and antibiotics.

DYSENTERIC POLYARTHRITIS

In bacterial dysentery a lesion of the joints, mostly of the knees, may be encountered in the period of recovery and sometimes one or two months after. For its clinical course it resembles rheumatoid polyarthritis and is of a toxicoallergic character. Sometimes an exudate forms in the joint cavity, in which case the disease takes a stubborn course.

In the acute period antidysenteric therapy is indicated (vaccination against dysentery, administration of sulpha drugs, antibiotics, etc.). Bed rest is necessary. Applications of dry heat to the affected joint and peroral administration of analgetics (analgine, etc.) and desensitizing preparations (pyramidon, antipyrine, aspirin) are prescribed. Ultraviolet light in erythema doses is recommended. Subacute and chronic forms are mainly treated by means of physiotherapy.

Prognosis is favourable in the majority of cases. Inadequate treatment may result in ankylosis of a joint and atrophy of the corresponding muscles.

In the acute period the *working capacity* is temporarily reduced, and in the presence of ankylosis and muscular atrophy the patient must be found a suitable occupation.

Prophylaxis is similar to that in dysentery; in addition, vigorous treatment of the latter disease is also necessary in order to avoid the development of polyarthritis.

DYSTROPHIC (NONSPECIFIC) ARTHRITIS ASSOCIATED WITH DISTURBANCE OF METABOLISM

Of the metabolic lesions of the joints the most important is gout described in the chapter on metabolic disorders (page 558).

Various disorders of the endocrine system cause lesions of the joints in which a substantial role is played by other factors, metabolic disorders in particular. The most frequent are lesions of the joints associated with a decreased or increased thyroid function and with ovarian hormonal deficiency, the so-called menopausal arthritis. A relatively less frequent occurrence is arthritis in affections of the parathyroid gland, in acromegaly, Itsenko-Cushing's syndrome, etc.

Dystrophic or noninfectious arthritides are sequels to hard physical work and overstrain of the joints. They occur in wrongly trained athletes and in persons frequently exposed to humid cold surroundings (house-painters, fishermen, laundry workers, operatives working in hot shops, etc.).

The disease begins insidiously, the joints become swollen, tender on movement, but no ankyloses or deformities develop, and the bones and cartilages remain unchanged. Upon a change in occupation the mobility of the joints is completely restored. Dystrophic arthritis is not attended with a rise in body temperature, leucocytosis, or an elevated ESR.

Primary deforming ankylosing osteoarthritis. The initial symptoms are pains followed by limitation of movements and deformities in the phalangeal joints and the wrists, ankles, elbows, and shoulders. An inflammatory exudate forms in the synovial membrane which later is invaded by granulation tissue as a result of which fibrosis and shrinkage of the joint capsule ensue. Similar changes occur in the periarticular tissue. In time the cartilage and bones undergo destruction; the cartilages are displaced by connective tissue and the bones become loose in structure with subsequent sclerosis. These changes in the bones and cartilages result in dislocations, subluxations, and ankylosis of the joints. No substantial changes occur in the blood count and the ESR (for treatment see under "Rheumatoid Arthritis").

Kashin-Beck or Urov disease (osteoarthritis deformans endemica). This disease is widespread in the Transbaikal area and other regions of Siberia, in the basin of the Urov River. It was for the first time described by Kashin and Beck whose names it bears to-day. The aetiology of this disease is uncertain. It is supposedly associated with contamination of cereals with poisonous fungi and a deficit of calcium, cobalt and copper and an excess of potassium, silicon and strontium in the soil.

The disease begins in childhood and adolescence. It affects the phalangeal joints of the hands, and later, the wrists, elbows, ankles, knees, and shoulders. The process is mainly localized to the bones. If the disease occurs in childhood the long bones cease to grow. Therefore the patient is dwarfish, his fingers are short, and the joints are destorted and markedly limited in movement. The thyroid gland is often enlarged.

The working capacity of such patients varies with the severity of changes in the joints. With first-degree changes the patient is incapacitated only partially; with second-degree changes he is unable to work, and with third-degree changes he is completely helpless.

Arthritis associated with endocrine disturbances. Menopausal and thyrotoxic arthritides are associated with dystrophy. Possibly a disturbance in the function of the endocrine glands only predisposes to rather than causes the development of arthritis. In these diseases deformities of the joints are also present.

Treatment is symptomatic.

TRAUMATIC ARTHRITIS

Not infrequently arthritis may be traumatic in character resulting from the effect of occupational factors (so-called static-traumatic arthritis). In occupations involving prolonged and great overexertion where safety rules are neglected the occurrence of such arthritis is inevitable. It should be noted that a number of factors contribute to the development of occupational arthritis, such as prolonged exposure to humid cold, dampness, and sharp temperature variations. A role is also played by chronic intoxication with alcohol, nicotine, etc. Occupational arthritis usually runs a benign course.

Treatment consists in elimination of unfavourable occupational factors. It also includes thermotherapy with paraffin, peat and ozocerite applications, local and general baths, diathermy, etc., as well as massage. Sulphur baths (36.6-36.2°C) are also given at a rate of 15-20 baths per course of treatment, preferably at a health resort.

Prognosis is usually favourable. Deformities of the joints and other complications are rare.

Prophylaxis consists in provision of favourable conditions in industry and strict observance of the rules of industrial hygiene.

HAEMOPHILIC ARTHRITIS

In haemophilia there are haemorrhages into the joint cavity, sometimes resulting from a slight trauma. Treatment of the underlying disease and the affected joint is necessary.

Prognosis is poor and in certain cases even ominous.

Prophylaxis consists in prevention of all factors likely to cause, a trauma. Special attention must be paid to the choice of occupation.

Lesions due to External Factors

THERMAL, CHEMICAL, AND ELECTRIC TRAUMAS

Burns. A burn is a damage to tissues as a result of exposure to a high temperature or chemical agents. Burns may be thermal or chemical depending on the causative factor. Thermal burns may be due to the action of flame, hot liquid, steam, as well as sunrays. The severity of a burn varies with the depth and size of the area involved. The most commonly used is a classification according to which burns are subdivided into three degrees.

A first-degree burn is characterized by redness and swelling at the site of affection. In a second-degree burn these symptoms are more pronounced, and, in addition, vesicles filled with a clear or cloudy fluid form in the affected area some time after the accident, sometimes within several hours. Third-degree burns are characterized, in addition to the above symptoms, by tissue necrosis.

Burns of the third as well as the second degree and even those of the first degree if they involve a large area of the skin, may cause severe general symptoms, such as derangements in the nervous system, the cardiovascular system, and in the parenchymatous organs, mainly the kidneys, changes in the blood, etc. Mortality from burns involving a large area is estimated to be at least 10 per cent on the average. First- and second-degree burns leave after healing a hardly noticeable or even quite imperceptible scar; third-degree burns leave ugly scars.

Local treatment of first-degree burns consists in applications of dressings of alcohol or a 2 per cent potassium permanganate solution. In second- and third-degree burns the damaged tissue is treated surgically. In burns of the third and as well as first and second degree, involving a large area of the body, measures must be taken above all against shock, then against intoxication, dehydration, progressive acidosis of tissues, and secondary infection.

Frostbites. Lesions due to exposure to cold are known as frostbites and may be manifested by general freezing, chilblain, and local frostbite. Frostbites are subdivided into four degrees. First-degree frostbites cause a circulatory disorder; second-degree, necrosis of the superficial layers of the skin; third-degree, complete necrosis of the skin and underlying layers; and fourth-degree, necrosis involving not only the integuments but also bones. It should be noted that in burns all manifestations are seen immediately after the accident, whereas in frostbites all processes develop slowly and the degree of damage is revealed gradually.

A person with a frostbite must be rapidly warmed and all measures should be taken to restore circulation of the blood in the affected parts and protect the latter against a secondary infection. In the presence of symptoms of second-degree frostbite treatment is based on surgical procedures.

Chemical burns. Chemical burns are due to exposure of the tissues to chemical agents having a caustic property, for example acids, alkali, as well as phosphorus and heavy metal salts. First aid in chemical burns consists in washing the damaged site with a liberal amount of water. In addition, acids are neutralized with alkali (2 per cent soda solution) and alkali with acids (2 per cent acetic or citric acid solution).

Injuries due to electric current. These injuries result from the action of atmospheric electricity (lightning) or electric current on the tissues. The higher the voltage, the greater the danger of electricity to human life. Electric current produces a thermal, chemical, and mechanical effect on tissues. In mild cases the general reaction to electric current is fright or fainting. In severe cases shock is encountered. In the majority of cases local lesions of the skin resulting from the action of electricity run a favourable course and require no special therapy. The principal therapeutic measure for severe general manifestations is artificial respiration which must be undertaken immediately since every minute of delay may cost the patient his life. Artificial respiration should be conducted patiently, sometimes for several hours. In addition, drugs stimulating respiration and the cardiovascular system must also be administered.

Sun and heat stroke. Prolonged exposure to sunrays or staying in a very hot room cause a disturbance in thermal regulation which results in a sun or heat stroke. Upon prolonged exposure to sunrays the local symptoms are first- and second-degree burns of the skin. General symptoms are a disturbance in respiration and in the cardiovascular function. There is fever, debility, torpor, and in severe cases the patient is unconscious. Convulsions and gastrointestinal disorders are also encountered sometimes.

A person who has sustained a stroke must be placed in a cool place and his tight clothes should be loosened. Further measures must be directed to supporting cardiovascular function and respiration and to preventing dehydration of the body (injections of camphor and caffeine, artificial respiration, introduction of a copious amount of liquid).

Research in nuclear physics and the discovery of the possibility of military and peaceful uses of atomic energy have confronted medicine with a number of problems involved in the effects of ionizing radiation (radiation in nuclear fission) on the human body.

It is perfectly clear that severe forms of radiation sickness and mass casualties are likely to be sustained during an atomic war. It must be borne in mind, however, that various forms of radiation sickness may occur in peacetime also. For example, there is a likelihood of any form of radiation sickness following errors in the application of x-ray therapy and as a result of overdosage of radioactive substances introduced for therapeutic or diagnostic purposes. Literature describes cases of acute radiation sickness after a breakdown of an atomic installation. The degree of severity of radiation sickness depends on a number of factors: the dose of ionizing radiation (the higher the dose the more severe the manifestations of the disease), the time of exposure to ionizing radiation, and the reactivity of the body. One and the same dose of ionizing radiation with an equal time of exposure may cause radiation sickness of varying severity. Correspondingly, chronic and acute forms of radiation sickness are distinguished clinically.

The chronic form of radiation sickness. The chronic form of radiation sickness runs a prolonged course and produces symptoms at a late period.

In this form a number of changes occur in all visceral organs which cause at first functional and later organic disturbances in the organs and physiologic systems. The chronic form of radiation sickness is known to develop in three stages.

The first stage is characterized by a number of transient symptoms evidencing the presence of only functional disturbances in the visceral organs. It should be remembered that in this period symptoms of the disease may be revealed only by comparison of the patient's condition before and after his exposure to the pathogenic factor.

The symptoms of the first stage of chronic radiation sickness are but slightly specific and are reversible. Among them are debility, general malaise, headaches, and insomnia. On objective examination symptoms of instability of the vegetative nervous system are discovered: sweating, vasomotor disorders, pronounced red dermographia, tremor of the fingers, etc. The cardiovascular system exhibits readily appearing dyspnoea, irregular pulse and arterial pressure with a tendency to hypotension. The gastrointestinal tract presents dyspeptic symptoms: anorexia, nausea, and eructation. The gastric secretion is unstable and is often reduced. Disturbances in the gastrointestinal tract are also manifested by a failure of its motor function.

Changes in the peripheral blood in the first stage of chronic radiation sickness are insignificant; there is a slight leucocytosis with lymphocytosis, and in more severe cases, leucopenia with lymphopenia. No changes occur in the function of the parenchymatous organs.

The second stage of radiation sickness is characterized by a gradual aggravation of the above-mentioned symptoms. This results from the transition of functional disorders into those associated with an organic lesion of a particular organ. In this stage, the patient develops multiple radiculoneuritis and a number of trophic changes appear in the skin: loss of elasticity, dermatitis which poorly responds to therapy, and local oedema. Organic gastric achylia develops. This results in aggravation of dyspeptic disturbances, with a further decrease or even complete loss of appetite. A disturbance in digestion associated with an organic lesion of the gastrointestinal tract as well as impairment of all forms of metabolism (protein, carbohydrate, fat) result in a weight loss.

The most severe manifestations of chronic radiation sickness occur in the third stage. This stage is characterized by a severe affection of the blood and vascular systems, endocrinic and metabolic disorders, as well as dysfunction of the central nervous system. An important place in the clinical picture of the disease is occupied by an intercurrent infection, at first local and later general (sepsis).

Changes in the blood system essentially consist in severe hypoplastic and even aplastic disorders in the haemopoietic organs. The most characteristic manifestation of this is marked leucopenia (from 4,000 to 2,000 and less leucocytes per 1 cu mm) with a prevailing drop in the number of lymphocytes.

Severe leucopenia leads to a diminution in the reactivity of the body, owing to which conditions are provided for the origination of septic foci at those sites where a considerable number of microbes (stomatitis, gingivitis, tonsillitis, etc.) are usually present.

The red blood exhibits a more or less marked anaemia, sometimes of the pernicious type (with megaloblastic type of haemopoiesis).

Among the characteristic signs of the third stage of radiation sickness are symptoms of haemorrhagic diathesis occurring in the form of haemorrhagic eruptions, bleeding of the gums, a positive tourniquet test, etc. Predisposition to bleeding in radiation sickness is conditioned by an increased permeability of the capillaries and a sharp reduction of the number of thrombocytes in the blood as a result of inhibition of their production in the bone marrow.

The body temperature is elevated, sometimes to a high level, which is partly due to an intercurrent infection and partly to a disorder in thermoregulation.

Changes in other visceral organs are not characteristic of radiation sickness. In general these changes may be described as dystrophic (dystrophic changes in the myocardium, vessels, liver, endocrine glands, etc.).

The acute form of radiation sickness. Acute radiation sickness results from exposure of the body to massive doses of ionizing radiation

A severe affection is caused by a dose of 100 to 300 rads. A 600 rad dose is considered lethal for man. Exposure to ionizing radiation in large doses is followed by substantial disturbances in the body.

The full clinical picture of acute radiation sickness does not develop immediately after exposure but appears after a certain period of time during which apparent signs of the disease may be either unmarked or absent altogether. In view of this the course of acute radiation sickness is conventionally subdivided into four periods following one another in succession.

The first period, or the period of primary reactions, begins within the early hours of exposure to ionizing radiation.

At this time the patient's condition resembles slight intoxication attended with nausea, vomiting, and sometimes with paraesthesia of the extremities. Attention is deserved by the fact that after a mass-scale exposure certain local symptoms may be observed even in this period at the site of the most intense irradiation.

The first period lasts from several hours to two days.

In the second, or latent, period the patient seems to be quite well. General disorders (a feeling of intoxication, dizziness, nausea, etc.), which were present in the first period, subside. A thorough examination of such patients, however, reveals insidious disturbances in the visceral organs, above all in the central nervous system and the blood. The latter exhibits a more or less pronounced leucopenia, mainly due to a reduction in the number of lymphocytes, and thrombopenia.

The length of the second period varies from several days to two weeks, after which the third period sets in. In severe cases, the second period may be absent.

In the third period a full clinical picture of radiation sickness is present. The patient's general condition becomes extremely grave. Symptoms of severe haemorrhagic diathesis, ulceronecrotic processes in the oral cavity and intestine develop, and dyspeptic disorders manifested by nausea and vomiting recur. Diarrhoea with a mucosanguineous discharge is encountered frequently. The temperature is elevated due to an intercurrent infection and a disturbance in thermoregulation. Considerable changes also occur in the blood system. In the period of manifest clinical symptoms of radiation sickness there is a progressive decline in the leucocyte count at first as a result of inhibition of lymphocytopoiesis and later of neutrophilic granulopoiesis. Simultaneously there occurs progressive thrombocytopenia to the degree of complete disappearance of thrombocytes from the peripheral blood. Later anaemia sets in also. Rapidly progressing anaemia is a symptom of a more severe affection and is an ominous prognostic sign.

Just as in chronic radiation sickness, in this period of the disease favourable conditions exist for the development of septico-infectious complications.

Septico-infectious complications of radiation sickness may begin in the intestine, the lungs, and other organs. They occur as a result of the saprophytic flora becoming pathogenic due to a reduced body resistance or activation of pre-existing foci of infection. The average duration of this period is about 30 days.

The fourth, or convalescent, period presents a gradual restoration of the disturbed functions of physiologic systems and organs. This period lasts for several weeks and even months. It is important to remember that complete restoration of the deranged functions may fail to occur.

The treatment of radiation sickness is a difficult and responsible task. It is of prime importance to know the specific cause of affection. Radiation sickness may develop as a result of exposure of the body to external radiation or to radioactive substances introduced into the body by one way or another.

In the latter case it is necessary to attempt to remove the radioactive substances from the body by means of diuretics, sudorifics, and purgatives.

Otherwise, therapeutic measures must be aimed at restoring the disturbed bodily functions. Very important in this connection are measures to control the changes occurring in the blood system. It is recommended to use for this purpose sodium nucleate, vitamins of the B complex (B_1, B_2, B_6, B_{12}), campolon, iron preparations, and blood transfusion. These remedies help to a certain extent to reduce the rapid aggravation of haematologic symptoms. A role of equal importance is played by measures against haemorrhagic diathesis. For this purpose, vitamin K and its preparations as well as calcium chloride are used.

The third important measure in the therapeutic complex is control of infection with antibiotics, and corresponding treatment of local foci (oral hygiene, etc.).

Great importance should be attached to symptomatic treatment which is conducted according to general rules.

Alimentary Toxicosis

Alimentary toxicosis is an acute disease due to ingestion of food containing pathogenic organisms and their toxins. It is characterized by a brief incubation period, develops simultaneously in a group of persons who have eaten one and the same food, and ceases after the latter has been withdrawn from use. An outbreak of food poisoning ends within a short period.

Alimentary intoxications are acute or less frequently chronic diseases resulting from ingestion of food containing nonbacterial poisons (lead, mercury, poisonous mushrooms, meat- or fish-decomposition products, weeds, etc.).

Chronic intoxications occur in systematic ingestion of small doses of poison.

BACTERIAL FOOD POISONING

This group of diseases is caused by bacteria of the genus *Salmonella*, organisms of botulism, staphylococci, streptococci, and other pathogenic organisms.

Food poisoning usually occurs upon ingestion of protein food (meat, fish, cottage cheese, milk) contaminated with pathogenic organisms or their toxins. This may take place in cases of violation of the rules of food storage, cooking and transportation. Bacteria and their toxins entering the gastrointestinal tract cause an inflammation and disturbance in the secretory, absorptive, and motor function. The antitoxic function of the liver is also disturbed, as a result of which poisonous substances enter the general blood flow and interfere with the functions of the central nervous system, the cardiovascular system, the kidneys, etc.

SALMONELLOSIS

In the majority of cases food poisoning is caused by bacteria of the genus *Salmonella*. Some of them exist as saprophytic organisms in the animal intestine and external environment and become pathogenic only under favourable conditions. Other *Salmonella* organisms, for example, *Salmonella typhimurium* and *Salmonella enteritidis* (Gärtner's bacillus) are pathogenic for man. Infection with *Salmonella* organisms results from a massive entry of living microbes of this group and their toxins into the human body.

The principal source of infection with salmonellae is the meat of animals and waterfowl, and eggs. The infection may be harboured by rodents infesting food industry establishments, kitchens, and food storages and contaminating food products with their excretions.

Clinical picture. Salmonellosis most commonly runs its course as a severe gastroenteritis or enterocolitis. The incubation period lasts from 5 to 24 hours. In mild cases the patient complains of abdominal pain, headache, and fever. The tongue is coated and dry. Only a mass-scale character of the infection makes it possible to differentiate between salmonellosis and common gastroenteritis. The clinical picture of moderately severe food poisoning essentially consists in an acute gastrointestinal disorder and fever. In severe forms of salmonellosis the patient complains of spasmatic abdominal pains, repeated vomiting, and diarrhoea; the stools are at first shaped and later become watery, often malodorous, with a green admixture and mucus. The patient complains of lassitude.The urinary secretion is reduced to the degree of anuria as a result of dehydration of the body; convulsions also occur. The tongue is dry and covered with a thick ashen-grey coating. Herpes appears sometimes on the lips. Palpation reveals tenderness in the umbilical region, rumbling in the caecum, or a spastically contracted sigmoid colon.

The pulse is frequent and weak, the arterial pressure is reduced. Collapse also occurs sometimes. The spleen and liver may be enlarged, with unmarked jaundice being present. Protein and erythrocytes are present in the urine. Neutrophilic leucocytosis is found in the blood. The disease runs its course with a normal and even subnormal temperature.

The course of salmonellosis, including its severe forms, is on the whole favourable and ends in complete recovery, but the development of collapse requires the most urgent therapeutic measures. Food poisoning is not infrequently followed by colitis, cholecystitis, and hepatitis.

Prophylaxis of salmonellosis consists in a rational organization of sanitary and veterinary inspection of slaughter livestock and meat as well as in measures to enforce sanitary rules in processing, manufacture, and storage of foodstuffs, finished and half-finished food products.

In all cases when bacteriologic examination is impossible meat must be used within 24 hours after its preliminary sterilization in autoclaves or by boiling in pieces of not more than 400 g in weight and 9 cm thick during $2^1/_2$ hours. If microbes are discovered only in the viscera the latter should be subjected to technical utilization and the carcasses boiled as described above. Upon discovery of salmonellae in the muscles and lymph nodes the latter are destroyed or utilized for technical purposes. For prophylaxis meat must be thoroughly boiled and stored at a temperature of not higher than 4°C because salmonellae are unable to reproduce in a cold environment. It is also recommended to use preserving agents—common salt, acetic acid, etc., for the same purposes.

Highly important for preventing the spread of salmonellosis is a strict sanitary inspection in the food industry, particularly detection of bacilli carriers. Accurate spacing of tables and boards for handling finished and raw foodstuffs, thorough washing and, whenever required, the use of chloramine for disinfection of kitchen tables may help safeguard heat-treated food products against infection with salmonellae from raw foodstuffs. Prophylaxis of salmonellosis consists in observance of certain hygienic rules (washing of the hands, disposal of refuse, particularly in summer when salmonellae reproduce intensively, destruction of flies which transmit infection, prohibition of drinking water from contaminated reservoirs, etc.).

BOTULISM

The first cases of the disease described in the 19th century were traced to ingestion of fish, sausages, and home-made vegetable and fruit preserves prepared without the use of substances destroying the causative agents of botulism and their spores. It has been found at present that botulism is caused not by one type of bacterium but by a group of related types A, B, C and E, of which the most common pathogenic varieties are A, B, C and partly E. In the Soviet Union types A, B and E have been encountered more frequently. These bacteria may develop only under anaerobic conditions. They carry spores which upon the death of the organism survive for a long time. Spores are highly resistant to chemical and physical factors.

The causal agent of botulism has its habitat in the soil from which it gains access to water and foodstuffs, and then to the human and animal intestine in which it propagates. Soil may be contaminated with human and animal faeces. Botulism is encountered upon ingestion of salted or smoked meat, ham, sausages, tinned vegetables, beans, maize, wheat, home-cooked fish. The causal agent of botulism intensively reproduces under anaerobic conditions in various foodstuffs at a temperature of 22-37°C. At a temperature of 10°C below zero its development and production of toxins are arrested. The vegetative forms of the organism are destroyed by their exposure to heat at 80°C during 10-16 minutes. Its toxin is destroyed in water at 100°C within 15 minutes.

Botulism develops as a result of the action produced on the body by a highly potent exotoxin formed in home-preserved foods. With a defect in hermetic sealing, however, infection may follow ingestion of factory-made tinned foods. In such cases, the "dormant" spores which have survived sterilization may be revitalized. After its entry into the stomach the toxin botulin is not destroyed, but rapidly causes paralysis of the motor nerves and paresis of the gastrointestinal tract. This results in retention of food in the stomach and increases intoxication.

Clinical picture. The disease sets in within several hours of ingestion of food. The incubation period lasts 17 to 24 hours but may be as short as 2 hours. The first symptoms of poisoning are paroxysmal abdominal pains, vomiting, diarrhoea, which may often be absent because of the rapid development of paresis of the gastrointestinal tract and the appearance of flatulence and constipation. The most severe changes occur in the central nervous system. The patient complains of diplopia and dimmed vision, thirst, and dryness in the mouth. Frequently, somnolence alternates with spells of excitement. The eyes are half-closed owing to paresis of the nerve innervating the upper lid and paralysis of the external and internal ocular muscles. Strabismus, nystagmus, and dilatation of the pupils are characteristic findings. The pulse is frequent, the arterial pressure is reduced, and the respiratory rhythm may be irregular. In certain cases there is choking and difficulty in deglutition. Paresis of the gastrointestinal tract results in constipation. The temperature is subfebrile but may also be normal or even subnormal.

The severity of the disease depends not only on the high virulence of the infection but also on the length of the incubation period. The longer the incubation period the greater the depressing effect of the toxin on the central nervous system. The disease lasts 4—8 days. Mortality may be as high as 70 per cent. Death ensues within the first 3-6 days, sometimes within 48 hours of the onset of the infection and is due to paralysis of the vasomotor nerves. Recovery is very slow, residual symptoms of paresis of the skeletal and smooth muscles being observed for a long time.

Prophylaxis of botulism consists in a strict sanitary inspection of foodstuffs, above all in the canning industry. The disease has become a rare occurrence in the Soviet Union owing to the implementation of sanitary and hygienic measures. In botulism outbreaks a specific antidote, an antitoxin, is employed for prophylactic purposes.

STAPHYLOCOCCAL FOOD POISONING

This disease is caused by pathogenic strains of staphylococcus which are widespread in nature, rapidly propagate at room temperature and as early as in 10-18 hours produce a toxin in many food products (cream, fancy cakes, dumplings, ice-cream, milk, tinned foods, ham, butter, etc.). The source of infection of foodstuffs is man and animals. Contamination of foodstuffs occurs when the latter are handled by persons with purulent skin lesions (paronychia, purulent wounds), tonsillitis, etc. Not infrequently the source of outbreaks of staphylococcal food poisoning is milk from cows afflicted with mastitis.

Clinical picture. The incubation period lasts not longer than 2-4 hours. The disease is characterized by brief nausea followed by frequent vomiting, abdominal pains, and sometimes diarrhoea. The pulse is frequent and weak. In severe cases there is a significant rise in temperature and convulsions, possibly with collapse. Sometimes the patient's condition becomes much better within 3-5 hours of the onset of the disease with recovery as early as in 24 hours.

Prophylaxis of staphylococcal food poisoning consists in veterinary control of milk cows and suspension of persons with purulent skin diseases and tonsillitis from their jobs involving contact with foodstuffs (personnel of public catering establishments, foodstores, etc.).

To prevent the reproduction of staphylococci milk and ice-cream mixtures are pausterized, and all creamy food (fancy cakes, etc.) are quickly sold out.

Treatment of food poisoning. The treatment of salmonellosis and staphylococcal food poisoning consists above all in measures to remove the casual agent and its metabolites from the gastrointestinal tract. The stomach is lavaged through a tube with 8-10 litres of water or a 2 per cent sodium bicarbonate solution. Immediately after this procedure 25-30 g of a saline purgative is given per os. In individual cases a high cleansing enema is administered, after which the patient is put into bed and hot-water bottles are applied to his feet and abdomen. In case of profuse and incessant vomiting one litre of physiologic solution is injected subcutaneously, sometimes 2-3 times in 24 hours. For convulsions 10 ml of a 10 per cent calcium chloride is administered intravenously; for a fall in the arterial pressure, caffeine, strychnine, camphor, ephedrine, and mesaton; for abdominal pains, subcutaneous atropine or platyphylline with papaverine. Other remedies are antibiotics and sulpha drugs given during 3-4 days (for salmonellosis).

On the first day of the disease the patient must abstain from food. He is allowed to have only strong tea and black coffee. From the second day on, thin gruels, rich broths, and soft-boiled eggs are given. At a later period, upon abatement of symptoms the patient may be maintained on the usual diet, excluding, however, milk, fatty meat dishes, tinned foods, piquant seasonings, and snacks for a certain time. Mineral water is recommended.

Treatment of botulism differs somewhat from that of other food poisoning. A role of crucial importance for the outcome of the disease is played by intramuscular injections of polyvalent botulinus antitoxin in a dose of 50,000-60,000 U. The first injection is made by Besredka's method. This antitoxin is efficacious even in severe cases of the disease. The stomach should be thoroughly lavaged with a 2 per cent sodium bicarbonate solution followed by a siphon enema since purgatives give a poor effect because of paresis of the intestine.

Poisoning

MUSHROOM POISONING

Poisoning with mushrooms of the *Helvellaceae* family containing helvellic acid is more often encountered in springtime. The latent period lasts not longer than 6-10 hours. The clinical symptoms are nausea, vomiting, intense abdominal pains, jaundice, enlargement of the liver and spleen, severe headaches, debility, delirium, convulsions, and coma. The mortality rate may be fairly high.

Poisoning with death cups (*Amanita phalloides*) is caused by phalloidin contained in the latter and is encountered in summer and particularly in autumn. The latent period lasts not longer than 12 hours. The clinical symptoms are tormenting abdominal pains, sometimes diarrhoea, persistent vomiting, intense thirst, dehydration of the body, chloropenia, convulsions, oliguria and cyanosis, at times enlargement of the liver, and jaundice. The mortality rate is high.

Poisoning with fly agaric (*Amanita muscaria*). Mushrooms of this genus contain muscarine, macoatropine and other toxins. Poisoning is encountered in summer and autumn and is due to ingestion of this mushroom mistaken for meadow mushroom or honey agaric. The latent period continues from 30 minutes to 24 hours.

The clinical symptoms are profuse perspiration, salivation, nausea, vomiting, diarrhoea, colicky pains, dizziness, hallucinations, and either dilation or narrowing of the pupils. The patient is somnolent, apathetic, and sometimes falls into coma. The mortality rate is not high.

Poisoning with mushrooms of the species *Lactarius piperatus, Lactarius vellereus, Paxillus involutus,* **and the genus** *Russulae* occurs all the year round upon ingestion of salted mushrooms inadequately treated and sorted. Gastroenteritis is a common clinical finding.

In mushroom poisoning the stomach is first of all lavaged with a 0.5 per cent tannin solution, and the patient is warmed. Mushroom poisoning due to muscarine is treated with atropine which is an antagonist of the latter. In addition, subcutaneous injections of agents stimulating cardiovascular function—caffeine, cordiamine, camphor, mesaton, etc.—are prescribed. In severe dehydration an abundant intake of liquid as well as intravenous and subcutaneous infusions of physiologic solution are recommended. Jaundice is treated with an intravenous infusion of a 40 per cent glucose solution half-an-hour after an injection of 8-12 U of insulin.

SOLANINE POISONING

Solanine is normally contained in potatoes; upon the latter's sprouting, however, its quantity is considerably increased. The clinical symptoms appear within 10-15 minutes of ingestion of sprouting potatoes as a scratching and burning sensation in the throat. Later nausea, vomiting, and sometimes diarrhoea occur. Cases of death have not been reported.

POISONING WITH BITTER FRUIT STONES

This poisoning commonly occurs within 4-5 hours of ingestion of bitter fruit stones (of apricots, peaches, almonds, cherries, etc.) which contain amygdalin. The latter is split by the enzyme emulsin into glucose, benzaldehyde, and hydrocyanic acid. The acid causes anoxaemia which brings about considerable changes in the body, the central nervous system in particular, because nerve cells are very sensitive to oxygen deficiency.

In mild cases the clinical symptoms are debility, dizziness, headaches, and nausea, which disappear within 4-6 hours. In severe cases the above symptoms are joined by diarrhoea, cyanosis, dyspnoea, clonic and tonic convulsions, slow respiration, and a feeling of constriction in the chest. The pulse may be weak and slow, the pupils are dilated, the mucous membranes and skin are bright-red in colour, and the patient's breath smells of almonds. Death occurs due to paralysis of the respiratory centre within several hours of the onset of the disease.

Treatment consists in repeated lavage of the stomach with a liberal quantity of a 0.05 per cent potassium permanganate or a 2 per cent hydrogen peroxide solution. Blood-letting, intravenous injection of a 10 per cent sodium hyposulphite solution, and inhalation of oxygen and amyl nitrite are necessary. Whenever required, artificial respiration, intravenous infusion of physiologic salt solution or glucose, injections of camphor, cordiamine, sympathol, etc., are given. The most reliable prophylactic measure is prohibition of sale of apricot and peach stones.

HENBANE POISONING

This poisoning is due to ingestion of home-baked bread of grain contaminated with henbane seed. The active poisonous substances are the alkaloids hyoscyamine and scopolamine contained in the seed of this plant. The patient complains of dryness in the mouth and throat, nausea, vomiting, difficult breathing, headaches, and dizziness. The pupils are dilated, the face is red, and tachycardia, intense excitement, and in severe cases delirium and hallucinations occur.

Upon the first signs of poisoning the stomach must be lavaged with a 0.05 per cent potassium permanganate or a 0.5 per cent tannin solution after which purgatives are administered. Excitement is reduced with morphine, omnopon, while collapse is treated with stimulating agents—cordiamine, ephedrine, caffeine, mesaton. Prophylaxis consists in a careful sorting of grain to remove weed seed.

BEAN FLOUR POISONING (FAVISM)

This condition is encountered upon ingestion of raw beans (*Vicia faba*) and flour prepared from them, since they contain toxic substances which are rendered harmless only by prolonged boiling. Adequate cooking of beans (soups, dishes of dried beans, etc.) prevents poisoning. The latent period of bean poisoning lasts 30-60 minutes. The clinical symptoms are nausea, vomiting, sometimes intense abdominal pains, and diarrhoea. These manifestations disappear within a few hours; however, severe cases of poisoning attended with haemoglobinuria, haemolytic jaundice, anuria, azotaemia, and other phenomena are also observed. To prevent bean poisoning the rules of heat treatment of bean flour used for preparing dishes which do not require prolonged heating should be observed strictly. Treatment is the same as that of other forms of food poisoning.

POISOINING WITH ANIMAL FOODS

Caviar, liver, and blood of certain fish (barbel, burbot, pike, perch) become toxic during the spawning season and may cause poisoning. Symptoms of poisoning appear soon after the ingestion of the above fish products. Mild cases of the disease resemble common gastroenteritis and enterocolitis. Severe cases are very much like cholera, especially after eating barbel caviar. Treatment is the same as in botulism.

ARSENIC POISONING

This poisoning usually follows indiscreet ingestion of arsenic-treated grain or bread baked from its flour. White arsenic (As_2O_3) as well as arsenic salts are widely used in the household and industry and therefore may be accidentally introduced into food products. Arsenous oxide, Paris green, and calcium arsenate are very often used for destruction of rodents and insects in the household, vegetable gardens and fruit orchards (sprinkling and dusting plants with arsenic agents). Arsenic poisoning causes a burning pain along the course of the alimentary tract and severe weakness in approximately 5 to 15 minutes. There are skin rashes, convulsions, facial oedema, and sometimes a chill with a rise in temperature (on the 2nd day of the disease). The patient is very much discomforted by sharp pains in the oesophagus and stomach, vomiting and cholera-like diarrhoea. The pulse is frequent and small, respiration is accelerated, and the face is pale and cyanotic. Sometimes the patient loses consciousness and sinks into a coma.

Arsenic poisoning is treated by repeated lavage of the stomach with lukewarm water to which magnesia usta has been added (20 g per litre of water) and emetics (apomorphine) are given. Vomiting is facilitated by drinking warm milk with whipped egg white and Antidotum arsenici (Ferri sulfurici oxydati soluti 100,0 + Aquae destillatae 300.0 and Magnesiae ustae 20.0 + Aquae destillatae 300.0) is administered. Both preparations are mixed immediately before use and taken per os by tablespoonfuls every 5-10 minutes; this mixture converts arsenic into poorly soluble compounds. If this antidote is unavailable the patient is given 50 g of magnesia usta, purgative salts, and diuretics. A hot-water bottle is applied to the abdomen. In collapse vasostimulants are prescribed.

COPPER AND ZINC POISONING

Copper poisoning follows the use of inadequately tinned kitchenware in which toxic compounds of soluble copper and fatty acids form upon contact with sour and fatty food. Zinc poisoning occurs upon ingestion of warm food cooked in zinc-coated pans and resembles copper poisoning in its clinical course.

Ingestion of the poisoned food is soon followed by the usual symptoms of gastroenteritis. The patient complains of intense thirst, a copper taste in the mouth, salivation, difficult respiration, sharp abdominal pains, headaches, muscular weakness, and burning pains along the oesophagus. Vomitus of blue-green masses, bloody diarrhoea, tenesmus, sometimes delirium and convulsions follow. The pulse is small and irregular, the arterial pressure is reduced, and urine secretion is diminished. The urine is of a black colour and contains much protein.

This poisoning is treated first of all with Antidotum metallorum— a saturated aqueous solution of hydrogen sulphide (100 ml of the solution contains 0.5-0.7 g of hydrogen sulphide, 0.1 g of sodium hydroxide, 0.38 g of magnesium sulphate and 1.25 g of sodium bicarbonate). The antidote forms insoluble and therefore nontoxic compounds with metals. The stomach is lavaged with a 0.1 per cent solution of potassium ferrocyanide, magnesia usta, or animal charcoal. Saline purgatives (but not castor oil) are administered per os. Abundant intake of liquid, including albumin water (egg white) is prescribed. Intakes of butter and milk are forbidden. In collapse cardiovascular stimulants, and in severe pains morphine, pantopon and promedole are administered.

LEAD POISONING

Lead and its compounds are widely used in industry and for household needs. Lead is handled by foundry workers, printers, tinsmiths, house-painters, etc. Lead compounds and metallic lead easily turn into dust and are introduced into the body through the respiratory tract and mouth. Lead poisoning sometimes results from a prolonged use of inadequately tinned or glazed kitchenware. When sour products (jam, marinade, pickles) are kept in such ware, lead salts may become soluble in quantities dangerous to health. From the respiratory tract and intestine lead enters the blood in which it circulates in the form of a salt of phosphoric acid and lead albuminate. With the blood lead is carried to the parenchymatous organs—the liver, spleen, and kidneys from where it enters the bones and is deposited in albuminate form, remaining there for years. Lead is excreted from the body through the kidneys and intestine. Unabsorbed lead and that excreted into the intestine is discharged through the latter. Lead may cause both acute and chronic intoxication. Apparently lead is a protoplasmatic poison affecting various tissues and organs.

Acute poisoning develops upon intoxication with large doses of lead compounds. The patient complains of a loss of appetite, debility, a metallic taste, and dryness of the mouth. The mucous membrane of the mouth and tongue is of a whitish colour. There is vomitus of ashen-grey masses, and black or blood-stained stools. The abdominal wall is indrawn, the pulse is tense. Convulsions, delirium, and coma are encountered sometimes.

Treatment. For acute poisoning magnesium sulphate, albumin water, milk, and mucilaginous broths are prescribed. In collapse vasostimulants are administered.

CHRONIC LEAD INTOXICATION

One of the most characteristic and severe symptoms of chronic lead intoxication is a specific clinical syndrome known as lead colic, which is due to spasm of the intestine caused by the action of lead on its smooth muscles.

Clinical picture. Lead colic is preceded by a 2-3 day prodromal period marked by dyspeptic symptoms (loss of appetite, nausea, vomiting, abdominal pains, constipation, less frequently diarrhoea) as well as muscular pains, arthralgia, and insomnia. These are followed by a rapid onset of severe paroxysmal pains in the abdomen which persist for several hours or for 2-3 days and longer. Pains located in the umbilical region are most characteristic. During an attack periods of abatement and aggravation of pain may alternate. In addition, vomiting, spastic constipation and retention of stools occur. The abdomen is indrawn, soft (sometimes, however, the abdominal muscles may exhibit a board-like rigidity), or may be considerably distended; rumbling and splashing sounds may be heard in the intestine.

Chronic lead intoxication disturbs the functions of parenchymatous organs, particularly often that of the liver. There is a tendency to hypertension which causes arteriolocapillary sclerosis with a predominant affection of the branches of the abdominal aorta. The skin is pale and slightly icteric. Jaundice is of a haemolytic type and is attended by bilirubinaemia and urobilinuria. Haemoglobin exchange is severely deranged. The urine contains a large amount of porphyrin pigments which lend it a bloody colour. Erythrocytes with basophilic granules and reticulocytes appear in the peripheral blood and hypochromic anaemia develops. Another important diagnostic finding is the so-called lead line on the gums, which is a narrow line of dark-grey or blue colour running along the gingival margins.

Treatment. Paranephral blocking is recommended. It is performed every other day until the cessation of colics. Blood transfusions in doses of 200 ml once in every 4-5 days, intravenous infusions of a 40 per cent glucose solution or physiologic salt solution, hot baths, and hot-water bottles are helpful. In mild cases spasmolytics—atropine, and platyphylline, and in severe cases, morphine, pantopon, and promedole are indicated. Polyneuritic pains are relieved with vitamin B_1 injections. In chronic intoxication the excretion of toxin is facilitated by a peroral administration of a 3 per cent potassium iodide solution.

Dietotherapy

The expenditures of energy necessary to maintain human life are recompensed by ingestion of food. The latter is absorbed upon digestion and is distributed to all organs and tissues. Part of the foodstuffs absorbed is utilized in the formation of cells, the remainder producing energy required for maintaining the vital activity of the body. The tissues and cells of the body consist of proteins, carbohydrates, fats, water, mineral salts and vitamins, which enter into the composition of food. Therefore, the substances consumed in the body may be replenished by intakes of food. The quantity of food to be taken by a healthy person in order to meet his energy requirements varies with his height, weight, the intensity of metabolic processes and the character of his occupation. It has been found that 30-40 Cal per kg of body weight are consumed in mental work, and from 50 to 60 Cal in physical work, depending on the degree of exertion. With weight being considerably less than normal in relation to height or, on the contrary, with an excessive weight the caloric intake is estimated on the basis of the standard normal rather than actual weight. For example, for a person measuring 170 cm in height and weighing 90 kg, the caloric intake is determined in relation to 70 kg because normal weight is estimated by deduction of 100 from the figure indicating height (170). The same pattern is followed in case the actual weight is below normal. Let us presume that the height is equal to 170 cm and the weight, to 50 kg. In this case the caloric requirements must also be calculated in relation to the weight of 70 kg. The diets of both healthy and sick persons must contain proteins, carbohydrates, fats, mineral salts, water and vitamins. A human being, however, cannot subsist proteins, fats or carbohydrates alone without detriment to his health. The amount of these nutrients must be in proportions required by a healthy person. Proteins (4.1 Cal per gram) are given in quantities of 90-100 g; fats (9.3 Cal per gram), 60-75 g, and carbohydrates (4.1 Cal per gram), from 450 to 500 g.

Proteins are plentiful in meat, fish, chickens, cottage cheese and cheese; fats, in butter, lard, sunflower oil, maize oil, cotton oil, cream, etc.; and carbohydrates, in sugar, bread, potatoes, cereals, etc. The diet for a healthy person includes common salt (10-15 g), calcium, potassium, iron and phosphorus salts, which are contained in large quantities in vegetables, meat, milk, and eggs. Vitamin A is present in many vegetables and fruits, milk, butter, caviar, cod-liver oil, etc.; vitamin B is contained in yeast, carrots, cabbage, beetroots, and grapes; vitamin B_2 (riboflavin) in yeast, eggs, meat, pulses, and milk. Vitamin D is present in considerable quantities in milk, eggs, butter, and particularly in cod-liver oil. Total or partial deficiency of this vitamin may cause rickets. Vitamin C (ascorbic acid) effective against scurvy is contained in vegetables, fruit, berries, particularly in black and red currants, sweetbrier, wild strawberries, etc. Vitamin K, increasing coagulation of the blood, is present in peas, tomatoes, cabbage, spinach, and other green plants.

A therapeutic diet is a powerful remedy in the treatment of many diseases. It is much different from the normal diet, since it is prescribed with due regard for the assimilability of food and for the patient's individual features and habits, and prohibits the intake of certain nutrients, restricts intakes of liquid and common salt, etc. The amount of solids and liquids eaten by a healthy person must not be larger than 1 kg for a meal. With regard to a sick person, however, this question is settled differentially, with reference to the character of the disease. For example, in pyloric stenosis, gastric and duodenal ulcer the amount of food eaten at a time must not exceed 200-300 g, which is necessary for a rapid evacuation of the stomach. The frequency of meals and length of intervals between them are also important. A healthy person must have at least three meals a day at 6-8 hour intervals. In certain diseases (pyloric stenosis, gastric and duodenal ulcer, colitis, diseases of the liver, the cardiovascular system, etc.) food should be taken in divided portions 5-6 times in 24 hours at 2-3 hour intervals. For this purpose, inmates of hospitals, sanatoria, etc., are mostly given four meals: breakfast at 8 (9) a.m., lunch at 11 a.m. (or at noon), dinner at 2 (3) p.m. and supper at 7 (8) p.m. In a number of diseases the patient is fed even more frequently—5-6 times daily.

In dietotherapy preference is given to those foodstuffs which contain the required quantities of nutritious proteins, carbohydrates, fats, mineral salts and vitamins. Particularly nourishing in this respect are milk and dairy products containing proteins (casein, albumin, globulin), readily soluble fats, and milk sugar. Milk is very wholesome as bland food causing no mechanical or chemical irritation of the gastric mucosa (peptic ulcer, gastritis with hypersecretion, etc.), in diseases of the kidneys and cardiovascular system, because it has dehydrating properties.

Of the dairy products used in diseases of the liver (hepatitis), diabetes, obesity, cottage cheese rich in methionine (a lipotropic substance), kefir and sour milk as laxatives for constipation are helpful.

Butter is a highly nourishing dairy product. It is easily absorbed and contains important vitamins. It is very useful for emaciating diseases and serves as a remedy inhibiting gastric hypersecretion (peptic ulcer, etc.). A similar food item is milk cream containing up to 20-25 per cent of fat. As distinct from butter it is easily tolerated, particularly when whipped.

Eggs are an important item of dietotherapy. However, they are poorly tolerated when eaten hard-boiled and sometimes even when fried. It is better to serve them soft-boiled or add them to steam-cooked dishes of meat, fish, vegetables, to soufflé and puddings, since they increase the porosity of the food thereby facilitating digestion.

Meat, particularly that of young animals, is widely used in dietotherapy; it contains a considerable amount of proteins and extractive substances. However, it is rich in connective tissue, especially in old animals; therefore a thorough cooking is necessary if this tissue is to be removed. This considerably facilitates the digestion of muscle tissue by enzymes. In this respect, chikens and lean sorts of fish (pike, pike perch, carp, etc.) are much more suitable because their muscle fibres are very tender.

Of the foodstuffs rich in carbohydrates wheat bread, flour foods, cereals eaten in gruels and soups and, especially, sugar and sweets which are easily assimilated and absorbed are widely used in dietotherapy.

An important food item is vegetables, which contain a considerable amount of liquid, cellulose, vitamins (carrots, beetroots, cauliflower, etc.), since after adequate cooking and especially in soufflé and purée form they are digested easily. Milk and eggs and butter are added to them when it is necessary to increase the caloric intake. Of vegetable foods cabbage, horse radishes and radishes are less easily tolerated.

Another important item in the dietotherapy is berries and fruits—apples, grapes, oranges, lemons, etc., as well as fruit juices. Fruits contain from 1 to 20 per cent fructose, grape sugar, aromatic substances, potassium, iron as well as vitamins and acids (malic, citric, etc.). Fruit and berries are extremely useful in the treatment of partial and total vitamin deficiency, in diseases of the liver (grapes), constipation, etc.

An increased intake of vitamins is necessary above all in partial and total vitamin deficiency. For example, in rickets there is greater need for calcium salts, which are contained in eggs, milk; in scurvy, for fresh vegetables, potatoes, fruit, berries, etc. Vitamins, however, are required not only in partial and total vitamin deficiency but also in many infectious diseases, anaemic states, gastrointestinal disorders attended with malabsorption or increased disintegration of vitamins.

An important role is played by rational cooking of food in order to preserve its vitamins. Vitamins A and C are destroyed by heat; therefore foodstuffs (fresh greens, berries, fruit, vegetables, etc.) containing these vitamins should be eaten raw or cooked at a low temperature. Butter should be added to food immediately before a meal. It is also important to cook food in closed saucepans without access of air so as to protect vitamins against destruction, as well as to eat fresh food since the latter's vitamin content is sharply reduced in long storage. For example, the amount of vitamin C in potatoes becomes negligible by springtime. Dried vegetables and fruit not subjected to special preliminary treatment are extremely poor in vitamins.

The liquid intake of a healthy person ranges from 1.5 to 2 litres in 24 hours. In a number of diseases, particularly of the kidneys and heart, which run their course with a circulatory disorder the liquid intake is severely restricted. In such cases liquid intake (including the liquid of soups, compotes, fruit, jellies, etc.) and urinary excretion should be measured every day. In inflammation of the urinary tract, diarrhoea, increased perspiration, intoxications, febrile diseases, abundant liquid intake is recommended, preferably tea with lemon, cranberry water, etc. The common salt intake is limited in diseases of the heart, kidney diseases with oedema, ascites, gastritis with hypersecretion, gastric and duodenal ulcer, and in obesity.

Some condiments such as pepper, vinegar, mustard, etc., as well as alcohol, rich broths, spices, roasted and smoked meat and fish, tinned foods, pickles are potent stimulants of juice secretion and irritants of the mucous membrane of the stomach; therefore, they must be, as a rule, excluded from a dietotherapy.

The diet should be prescribed according to the character of the disease, the patient's individual characteristics, degree of nourishment, and gastrointestinal function.

The following types of diet are used: (a) mechanically and chemically bland; (b) corrective or reconstructive; (c) substitutive. They are described in detail below.

A mechanically and chemically bland diet excludes all mechanical (animal and vegetable cellular tissue) and chemical (broths, smoked and roasted foods, tallow fats, pickles, spices, vinegar, alcohol, coffee, etc.) irritants. In this diet meat, fish, chickens, vegetables, carbohydrates (cereals) are used in steam-cooked and purée form while intakes of butter, cream, and milk are increased because they are mild stimulants of gastrointestinal secretion. It should be borne in mind, however, that milk is sometimes tolerated very poorly by patients with inflammatory changes in the intestine (colitis). A bland diet excluding mechanical and chemical irritants is prescribed for patients with peptic ulcer, gastritis attended with hypersecretion, colitis, diseases of the liver and bile ducts. In the latter case, however, restrictions on intakes of butter (50 g) and proteins are necessary in view of a possible overload of protein decomposition products on the liver.

A corrective diet is prescribed when it is necessary to increase or reduce the intake of some nutrients at the expense of others or increase the standard food intake. For example, in emaciation the intake of highly nourishing foods (fats) is increased at the expense of proteins and, on the contrary, in obesity the intake of fats and carbohydrates is sharply decreased by increasing the protein intake. In diseases of the liver carbohydrate intake is increased in order to enlarge the amount of glycogen deposited in the liver and intensify hepatic antitoxic function, while the intake of proteins is reduced. Protein intake is diminished in the diet for patients with an impaired nitrogen-excreting function of the kidneys in order to eliminate protein decomposition products from the body. And conversely, it is recommended to ingest a considerable amount of proteins in conditions involving an intensified consumption of nutrients—in infectious diseases, lipoid nephritis, dystrophic phases, and in disorders of the circulation. Restrictions on fats are indicated in obesity, pancreatitis, atherosclerosis, and diarrhoea. A diet rich in lipotropic (methionine) substances (cottage cheese, oatmeals, milk, etc.) is prescribed for fatty degeneration of the body in order to deplete the latter's reserves of fat and replace them with glycogen.

A substitutive diet is prescribed not only for partial or total vitamin deficiency but also for a number of diseases in which it is necessary to supply an additional amount of food rich in readily soluble fats, nutritious proteins and amino acids, mineral substances (phosphates, iron, calcium, etc.), for undernourishment, a dietary deficiency, anaemic states, etc.

The so-called "zig-zag" method is also used in dietotherapy. It essentially consists in that the diet prescribed during the given disease is combined with the so-called "high-intake" days in which the earlier excluded dishes are served at first once weekly, and later more frequently, along with the principal bland diet. These "zigzags" prevent the health hazards of a strict and monotonous dietary regimen which may cause a considerable deficit of valuable nutrients in the body.

Because it is impracticable to institute an individual diet for any particular patient a group diet proposed by M. Pevzner is extensively used in the Soviet Union.

There are fifteen therapeutic diets. Diets No. 1*a*, No. 1*b* and No. 1 are prescribed for gastric and duodenal ulcer and gastritis with hypersecretion. The dishes included in the above diets contain no mechanical and chemical irritants. Diet No. 1*a* consists of milk, cream, butter, mucilaginous soups (of rice, pearl-barley, semolina), eggs, non-sour jellies, sweet dishes, vegetable and fruit juices. Diet No. *1b* differs from menu No. *1a* in having, in addition to 50 g of white rusks, puréed gruels with butter, meat or fish quenelles. Diet No. 1, in addition to dishes listed in diets No. *1a* and No. *1b* includes stale wheat bread, steam-cooked cutlets, boiled lean meat and fish (pike perch, pike, perch), steam-cooked omelettes and souffle, boiled puréed vegetables and fruit, weak tea with milk or cream. In acute periods of the disease as well as in bleeding, treatment is begun with diet No. *1a*, usually changing over to diet No. *1b* in a fortnight, and to diet No. 1 in another fortnight. The latter diet is continued for 3 months and longer. Patients with peptic ulcer and gastritis receive food every 2-3 hours, the intake of salt being strictly limited.

Diet No. 2 is prescribed for gastritis with hyposecretion and is mostly mechanically bland. It includes the principal nutrients required by a healthy person but they are served in purée or minced form, excluding rye bread, as well as fatty sorts of meat, fish, lard, smoked foods, etc.

Diet No. 3 is also prescribed for constipations and is rich in vegetable cellulose. Diet No. 4 is used for acute inflammations in the intestine; it is chemically and mechanically bland and, in addition, does not contain milk, since the latter causes a sharp relapse of the disease.

Diet No. 5 is prescribed for acute diseases of the liver, bile ducts, gallbladder, and consists mainly of milk and vegetables. All dishes are served in purée form. Lean meat or boiled and puréed fish is served once daily, the intake of butter being strictly limited. Wheat stale bread is also given. The diet excludes spices and extractive substances, piquant dishes, sausages, smoked foods, piquant cheese, meat, fish and mushroom soups, eggs, tomatoes, spinach and sorrel.

Diet No. 6 is used for uric acid diathesis and gout. Peas, spinach, sorrel and radishes must be avoided. Intakes of vegetable and fruit juices in a liberal amount are recommended.

Diet No. 7 is intended for the treatment of kidney patients. Extractive substances are excluded, protein intake is limited, especially in azotaemic uraemia, with severe restrictions on salt and liquid. Diet No. 7 consists of flour and sweet dishes, butter, milk, berries, boiled meat, fish, chickens, fruit, fruit juices, vegetables, excluding horse radish, radish, garlic, parsley, onions, and spinach. The patient is given 2 g of salt, while wheat unsalted bread is specially baked for kidney patients.

Diet No. 8 is prescribed for obesity and consists of rye bread, vegetables, fruit, berries, lean meat, fish, chickens, cottage cheese, milk and kefir. The food also contains a large amount of vegetable cellulose. Intakes of fat, flour and sweet dishes and sugar are strictly limited.

Diet No. 9 is individual, and is intended for patients with diabetes mellitus.

Diet No. 10 is prescribed for cardiovascular diseases. Intakes of liquid and salt are restricted, while all piquant dishes, smoked foods, snacks, coarse cellulose, vegetables and fruits are excluded. Meat and fish are served in purée form, and wheat bread is given. Milk and vegetable soups, berry, fruit and vegetable juices, compotes and thin jellies are recommended.

Diets Nos. 11, 12, 13, 14 are used comparatively rarely. Diet No. 11 is prescribed for emaciated patients, those with pulmonary tuberculosis. Diet No. 12 is prescribed for diseases of the nervous system, mostly functional. Diet No. 13 is used after operations on the abdominal cavity. Diet No. 14 is prescribed for phosphaturia and oxaluria, with renal calculi. At the same time intakes of vegetables, greens, fruit, dairy products and eggs are restricted. Meat, fish and flour dishes are the principal items in the diet.

Diet No. 15 is a standard rationale, which is prescribed for patients not requiring special dietotherapy.

After a course of dietotherapy many patients require a strict observance of the dietary regimen during a certain period. For this purpose, special dietatic dining-rooms have been set up in the Soviet Union. A course of treatment in these establishments lasts 1-2 months, and in case of special indications (diabetes, chronic nephritis, peptic ulcer, etc.), several months. The diet is drawn up by the diet nurse after she has obtained from the ward nurse the required information on the number of patients and their respective diets prescribed by the physician. In drawing up the menu the diet nurse should take account of its caloric value, including the required intakes of fats, carbohydrates, proteins and vitamins. She should see to it that even a strictly defined diet is sufficiently diversified. The patient must not be given one and the same gruel every day, nor compotes or jellies prepared from the same berries and fruit. A patient maintained on a diet usually has a poor appetite and a monotonous diet is likely to cause a total loss of appetite.

The ward nurse keeps a check on the adequacy of food items listed in the menu. She should also take care that meals are served hot and, whenever necessary, warmed as prescribed by the physician.

During meals the nurse must mostly be present in the ward for bed-ridden patients who can help themselves with their meals. Debilitated patients and those for whom all motions are forbidden are fed by the nurse.

Her duty is to ensure that food is served at appointed hours in accordance with the menu, and to take care that patients do not eat forbidden food brought by their visitors. A menu sheet must be posted in the dining-room every day.

FORMULAS

1. Cardiovascular agents

No. 1

Rp. Pulv. fol. Digitalis 0.05
Sacchari albi 0.3
M.f. pulv. D.t.d. N. 15
S. One powder 3 times daily

No. 2

Rp. Pulv. fol. Digitalis 0.1
Butyri Cacao 2.0
M.f. suppos. D.t.d. N. 9
S. One suppository 3 times daily

No. 3

Rp. Inf. fol. Digitalis
ex 0.6 : 200.0
S. One tablespoonful 3 times daily

No. 4

Rp. Inf. fol. Digitalis ex 1.0 : 200.0
DS. For enema in 40.0
in the morning and at bedtime

No. 5

Rp. Pulv. fol. Digitalis 0.05
Chinidini 0.05
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 6

Rp. Pulv. fol. Digitalis 0.05
Chinini bromati 0.05
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 7

Rp. Digalen-neo 1.0
D.t.d. N. 6 in amp.
S. One ml for subcutaneous injection
1-2 times daily

No. 8

Rp. Inf. herbae Adonidis vernalis 6.0 : 200.0
DS. One tablespoonful 3-4 times daily

No. 9

Rp. Adonileni 15.0
DS. 7-10 drops 3 times daily

No. 10

Rp. Adonisidi 20.0
DS. 15 drops 3 times daily

No. 11

Rp. T-rae Strophanthi 10.0
DS. 5 drops 3 times daily

No. 12

Rp. Sol. Strophanthini 0.05%-1.0
D.t.d. N. 10 in amp.
S. 0.25-0.5 ml intravenously
in 20 ml of 40 per cent glucose solution
(inject slowly during 3-5 min)

No. 13

Rp. Sol. Corglyconi 0.06%-1.0
D.t.d. N. 10 in amp.
S. 0.5-1 ml intravenously
in 20 ml of 40 per cent glucose solution

No. 14

Rp. Validoli 10.0
DS. 5 drops for pains (on sugar)

No. 15

Rp. Sol Nitroglycerini alcoholicae 1% 5.0
DS. 1-2 drops on lump of sugar
for pains (under tongue)

No. 15*a*

Rp. Nitroglycerini 0.0005
D.t.d. N. 12 in tabul.
S. 1 tablet for pains (under tongue)

No. 16

Rp. Nitranoli 0.002
D.t.d. N. 20 in tabul.
S. One tablet 3-4 times daily after meals

No. 16*a*

Rp. Nitropentoni 0.01
D.t.d. N. 50 in tabul.
S. One tablet 3-4 times daily
(under tongue)

No. 16*b*

Rp. Tipheni 0.02
D.t.d. N. 40 in tabul.
S. One tablet 3 times daily

No. 17

Rp. Sol. Natrii nitrosi 1.0 : 200.0
DS. One tablespoonful 3 times daily

No. 18

Rp. Amylii nitrosi 0.5
D.t.d. N. 3 in amp.
S. Moist handkerchief with 2-3 drops
and inhale

No. 19

Rp. Novocain-amidi 0.5-1.0
D.t.d. N. 12 in tabul.
S. One tablet 3-4 times daily

No. 20

Rp. Dibazoli 0.02
Sacchari 0.3
M.f. pulv. D.t.d. N. 12
S. Two powders 3 times daily

No. 21

Rp. Dibazoli
Papaverini h.āā 0.02
Diuretini 0.3
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 22

Rp. Sol. Dibazoli 2-1% 1.0
D.t.d. N. 6 in amp.
DS. 1-2 ml intramuscularly or
intravenously

No. 23

Rp. Diuretini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 24

Rp. Papaverini h. 0.04
Euphyllini 0.2
Luminali 0.02
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 25

Rp. Papaverini 0.02
Luminali 0.03
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 26

Rp. Euphyllini 0.3
Butyri Cacao 2.0
M.f. suppos. D.t.d. N. 12
S. One suppository 3 times daily

No. 26*a*

Rp. Euphyllini 0.2
Papaverini h. 0.02
Dimedroli 0.03
M.f. pulv. D.t.d. N. 12
S. One powder 2-3 times daily

No. 27

Rp. Sol. Euphyllini 2.4% 10 ml
D.t.d. N. 10 in amp.
S. One ampule in 40% glucose solution
(20 ml) intravenously 1-2 times
daily (inject very slowly!)

No. 28

Rp. Chinidini 0.1
D.t.d. N. 12
S. One powder 3 times daily

No. 29

Rp. Neodicumarini 0.15-0.2
Sacchari 0.2
M.f. pulv. D.t.d. N. 12
S. One powder 2-3 times daily
(check on blood prothrombin)

No. 29*a*

Rp. Phenylini
in tablets of 0.02-0.03
D.t.d. N. 12
S. One tablet 2-3 times daily
(check on blood prothrombin)

No. 29*b*

Rp. Pelentani 0.15
D.t.d. in tabul. N. 10
S. One tablet 2-3 times daily
(check on blood prothrombin)

No. 30

Rp. Sol. Ephedrini hydrochlorici 5% 1.0
D.t.d. N. 10 in amp.
S. 0.5-1.0 ml 1-2 times a day subcutaneously

No. 31

Rp. Ol. Camphorati 20% 2.0
D.t.d. N. 10 in amp.
S. 2 ml 3-6 times a day subcutaneously

No. 32

Rp. Coffeini natrio-benzoici 0.2-0.1
D.t.d. N. 12
S. One powder 2 times daily

No. 33

Rp. Sol. Coffeini natrio-benzoici 10% 1.0
D.t.d. N. 10 in amp.
S. One ampule 2 times
a day subcutaneously

No. 34

Rp. Cordiamini 1.0
D.t.d. N. 10 in amp.
S. One ampule 2-3 times a day
subcutaneously

No. 35

Rp. Sol. Atropini sulfurici 0.1% 1.0
D.t.d. N. 10 in amp.
S. 0.5-1.0 ml 1-2 times a day
subcutaneously

No. 35*a*

Rp. Sol. Mesatoni 1% 1.0
D.t.d. N. 10 in amp.
S. 0.5-1.0 ml subcutaneously

No. 35*b*

Rp. Mesatoni 0.01
Sacchari albi 0.2
M.f.p. D.t.d. N. 10
S. One powder 2 times daily

No. 36

Rp. Sol. Adrenalini hydrochlorici 0.1% 1.0
D.t.d. N. 10 in amp.
S. 0.25-0.5-1.0 ml subcutaneously

No. 37

Rp. Sol. Strychnini nitrici 0.1% 1.0
D.t.d. N. 10 in amp.
S. 1 ml 1-3 times a day subcutaneously

No. 38

Rp. Sol. Corazoli 10% 1.0
D.t.d. N. 10 in amp.
S. 1 ml 1-2 times a day
subcutaneously or intravenously

No. 39

Rp. Camphorae monobromatae 0.3
D.t.d. N. 12 in tabul.
S. One tablet 3 times daily

No. 40

Rp. Camphorae tritae 2.0
T-rae Valerianae aethereae 20.0
MDS. 20 drops 3 times daily

No. 41

Rp. T-rae Strychni 10.0
DS. 8 drops 3 times daily

No. 42

Rp. Sol. Glucosae 40% 20.0
D.t.d. N. 6 in amp.
S. 1 ampule intravenously

No. 42*a*

Rp. Rutini 0.02
D.t.d. N. 12 in tabul.
S. 1-2 tablets 3 times daily

II. Diuretics

No. 43

Rp. Novuriti 1.0
D.t.d. N. 10 in amp.
S. 0.5-1.0 ml intramuscularly

No. 43*a*

Rp. Fonuriti 0.25
D.t.d. N. 12 in tabul.
S. One tablet 1-2 times daily

No. 44

Rp. Ammonii chlorati 0.5
D.t.d. N. 12 in caps.
S. One capsule 4 times daily

No. 45

Rp. Sol. Ammonii chlorati 5% 200.0
DS. One tablespoonful 3-5 times daily

No. 46

Rp. Diuretini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 46*a*

Rp. Hypothiazidi 0.025
D.t.d. in tabul. N. 20
S. 1-2 tablets 2 times daily

No. 47

Rp. Urotropini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 48

Rp. Sol. Urotropini 40% 10.0
D.t.d. N. 10 in amp.
S. One ampule intravenously

No. 49

Rp. Kalii acetici 20.0
Aq. destill. 200.0
MDS. One tablespoonful 4 times daily

No. 50

Rp. Decocti fol. Uvae ursi 10.0 : 200.0
DS. One tablespoonful 5-6 times daily

No. 51

Rp. Ureae puri 10.0
in charta cerata
D.t.d. N. 12
S. 1 powder 3 times daily

No. 52

Rp. Saloli 0.5
Extr. Belladonnae 0.015
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 53

Rp. Salitropini 5.0
D.t.d. N. 10 in amp.
S. One ampule intravenously

No. 54

Rp. Sol. Calcii chlorati 20 : 200.0
DS. One tablespoonful 3 times daily

No. 54*a*

Rp. Kalii chlorati 10.0
Aq. destill. 200.0
S. One tablespoonful 3-4 times daily

No. 55

Rp. Theobromini 0.3
D.t.d. N. 10
S. One powder 3 times daily

III. Agents Used in Pulmonary Diseases and Expectorants

No. 56

Rp. Codeini puri 0.015
Sacchari 0.3
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 57

Rp. Codeini phosphorici 0.015
Sacchari 0.2
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 58

Rp. Dionini 0.01
D.t.d. N. 12 in tabul.
S. One tablet 3 times daily

No. 59

Rp. Sol. Dionini 1% 10.0
S. 5-8 drops 2 times daily

No. 60

Rp. Inf. herbae Thermopsidis 0.6 : 200.0
S. One tablespoonful 3 times daily

No. 61

Rp. Inf. rad. Ipecacuanhae 0.6 : 200.0
Natrii bicarbonici 4.0
MDS. One tablespoonful 3 times daily

No. 62

Rp. Kreosoti 15.0
DS. Five drops 3 times daily

No. 63

Rp. Heroini 0.003
Guajacoli carbonici 0.3
D.t.d. N. 12
S. One powder 3 times daily

No. 64

Rp. Terpini hydrati 0.3
D.t.d. N. 12
S. One powder 3 times daily

No. 65

Rp. Codeini puri 0.015
Terpini hydrati 0.3
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 66

Rp. Asthmatoli 1 box
DS. For smoking during attack of asthma

No. 67

Rp. Ephedrini hydrochlorici 0.025
Sacchari 0.3
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 68

Rp. Sol. Natrii benzoici 15% 10.0
D.t.d. N. 10 in amp.
S. For intravenous infusion

No. 69

Rp. Sol. Natrii chlorati 10% 20.0
D.t.d. N. 10 in amp.
S. For intravenous infusion

No. 70

Rp. Kalii jodati 6.0
Aq. destill. 200.0
DS. One tablespoonful 3 times daily

No. 71

Rp. Heroini 0.005
Sacchari 0.3
M.f. pulv. D.t.d. N. 10
S. One powder 3 times daily

No. 72

Rp. Sol. Lobelini hydrochlorici 1% 1.0
D.t.d. N. 6 in amp.
S. 0.3-0.5 ml subcutaneously

IV. Purgatives and Cholagogic Agents

No. 73

Rp. T-rae Amarae 20.0
DS. 20 drops 3 times daily before meals

No. 74

Rp. Extr. fluidi Condurango 20.0
DS. 15 drops 3 times daily

No. 74*a*

Rp. T-rae Absinthii 10.0
DS. 10-15 drops 3 times daily half-an-hour before meals

No. 75

Rp. Pancreatini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 76

Rp. Acidi muriatici diluti 15.0
DS. 15 drops per quarter-glassful
of water 3 times daily at meals

No. 77

Rp. Pepsini 4.0
Acidi muriatici diluti 6.0
Aq. destill. 200.0
MDS. One tablespoonful 3 times
daily at meals

No. 78

Rp. Magnesiae ustae 0.5
Extr. Belladonnae 0.015
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 79

Rp. Magnesiae carbonici 0.5
Extr. Belladonnae 0.015
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 80

Rp. Carboleni 1.0
D.t.d. N. 12
S. One powder 4 times daily

No. 81

Rp. Bolus albae 1.0
D.t.d. N. 12
S. One powder 3 times daily

No. 82

Rp. Extr. Belladonnae 0.015
Butyri Cacao 2.0
M.f. suppos. D.t.d. N. 12
S. One suppository into anus

No. 83

Rp. Anaesthesini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 84

Rp. Sol. Apomorphini hydrochlorici 1% 1.0
D.t.d. N. 6 in amp.
S. $^{1}/_{2}$-1 ampule subcutaneously

No. 85

Rp. Rad. Rhei pulverati 0.5
D.t.d. N. 6
S. One powder 3 times daily

No. 86

Rp. Magnesiae sulfurici 25.0
DS. Dissolve in half-glassful of water
for one intake

No. 87

Rp. Sol. Magnesiae sulfurici 20-25% 10.0
D.t.d. N. 3 in amp.
S. 10 ml intravenously and intramuscularly

No. 88

Rp. Salis Carlsbadensis 50.0
DS. Half-teaspoonful in half-glassful
of water on empty stomach

No. 89

Rp. Benzonaphtholi 0.3
D.t.d. N. 12
S. One powder 3 times daily

No. 90

Rp. Extr. Belladonnae 0.015
Papaverini hydrochlorici 0.02
Sacchari 0.3
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 91

Rp. Tabul. Phenolphthaleini 0.1
D.t.d. N. 12
S. One tablet at bedtime

No. 92

Rp. Purgeni 0.1
D.t.d. N. 6 in tabul.
S. 1-2 tablets per intake

No. 93

Rp. Platyphyllini bitartarici 0.005
Sacchari 0.2
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 94

Rp. Calcii Carbonici 0.5
D.t.d. N. 15
S. One powder 3 times daily

V. Anthelmintic Agents

No. 95

Rp. Santonini 0.1
D.t.d. N. 12
S. One powder 3 times daily

No. 96

Rp. Methylenblau medicinali 0.1
D.t.d. N. 6 in caps.
S. One capsule 3 times daily after meals during 2-3 days

No. 96*a*

Rp. Sulfuris praecipitati 1.0
D.t.d. N. 15
S. One powder 3 times daily at meals for 5 days running

No. 97

Rp. Heptylresorcini 0.1
D.t.d. N. 12 in tabul.
S. 2 tablets during 1 hour at 5-minute intervals on empty stomach

No. 98

Rp. Osarsoli 0.25
D.t.d. N. 20 in tabul.
S. One tablet 4 times daily

No. 98*a*

Rp. Thymoli 1.0
D.t.d. N. 6 in capsul. gelat.
S. One capsule on empty stomach

No. 99

Rp. Sancapheni
in tabul. orig. N. 16
S. 4 tablets on empty stomach after tea and piece of bread, another 4 tablets in 30 minutes, 1 tablet of purgen in the evening. Repeat this procedure on the next day

No. 100

Rp. Extr. Filicis maris 0.5
D.t.d. N. 12 in capsul. gelat.
S. One capsule every 5 minutes
during 30-40 minutes

No. 101

Rp. Phenothiazini 0.5
D.t.d. N. 15
S. One powder 3 times daily

VI. Antirheumatic, Antipyretic, Tranquillizing and Soporific Agents

No. 102

Rp. Sol. Morphini muriatici 1% 1.0
D.t.d. N. 10 in amp.
S. For subcutaneous injection

No. 103

Rp. Sol. Pantoponi 1% (2%) 1.0
D.t.d. N. 12 in amp.
S. For subcutaneous injection

No. 104

Rp. Sol. Omnoponi 1% 1.0
D.t.d. N. 10 in amp.
S. For subcutaneous injection

No. 105

Rp. Sol. Promedoli 1% 1.0
D.t.d. N. 10 in amp.
S. One ampule subcutaneously

No. 106

Rp. Morphini muriatici 0.01
Sacchari 0.3
D.t.d. N. 10
S. One powder for pains

No. 107

Rp. Pantoponi 0.01
Sacchari 0.3
D.t.d. N. 12
S. One powder for pains

No. 108

Rp. Veronali 0.3
D.t.d. N. 10
S. One powder at bedtime

No. 109

Rp. Medinali 0.3
D.t.d. N. 10
S. One powder at bedtime

No. 110

Rp. Luminali 0.1
D.t.d. N. 12
S. One powder at bedtime

No. 111

Rp. Barbamyli 0.2
D.t.d. N. 6
S. One powder at bedtime

No. 112

Rp. Nembutali 0.1
D.t.d. N. 6
S. One powder at bedtime

No. 113

Rp. T-rae Leonuri 30.0
S. 20 drops 3 times daily

No. 114

Rp. Sol. Natrii bromati 6.0 : 200.0
DS. One tablespoonful 3 times daily

No. 115

Rp. Sol. Natrii bromati 10% 10.0
D.t.d. N. 3 in amp.
S. 5-10 ml intravenously

No. 116

Rp. Inf. rad. Valerianae 6.0 : 200.0
Natrii bromati 2.0
Kalii bromati 2.0
MDS. One tablespoonful 3 times daily

No. 117

Rp. Analgini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 118

Rp. Inf. herbae Adonidis vernalis 6.0 : 200.0
Natrii bromati 6.0
Codeini phosphorici 0.2
MDS. One tablespoonful 3 times daily

No. 119

Rp. Pyramidoni 0.25
Coffeini puri 0.05
M.f. pulv. D.t.d. N. 6
S. One powder 3 times daily

No. 120

Rp. Chlorali hydrati 2.0
Aq. destill. 100.0
DS. 50.0 per enema 1-2 times daily

No. 121

Rp. Natrii salicylici 1.0
D.t.d. N. 20
S. One powder at 3-hour intervals

No. 122

Rp. Aspirini 0.5
D.t.d. N. 12
S. One powder 6 times daily

No. 123

Rp. Pyramidoni 0.5
D.t.d. N. 12
S. One powder 3-4 times daily

No. 124

Rp. Acesali 1.0
D.t.d. N. 12
S. One powder 3-6 times daily

No. 125

Rp. Sol. Natrii salicylici 10% 10.0
Sterilisetur!
DS. For intravenous injections

No. 126

Rp. Antipyrini 0.5
D.t.d. N. 12
S. One powder 3 times daily

No. 127

Rp. Natrii salicylici 10.0
Aq. destill. 200.0
MDS. One tablespoonful 6-8 times daily

No. 128

Rp. Atophani 1.0 (0.5)
D.t.d. N. 10
S. One powder 3 times daily
in half-glassful of mineral water
during 5 days to be followed
by five-day interval

VII. Haemostatic and Antianaemic Agents, Agents for Diseases of the Haemopoietic System

No. 129

Rp. Sol. Calcii chlorati 10% 10.0
D.t.d. N. 6 in amp.
DS. 5-10 ml intravenously.

No. 130

Rp. Sol. Natrii chlorati 10% 20.0
D.t.d. N. 6 in amp.
DS. 5-10 ml intravenously

No. 131

Rp. Sol. Arsenicalis Fowleri 20.0
DS. 8-15 drops 3 times daily

No. 132

Rp. Urethani 1.0
D.t.d. N. 6 in caps. gelat.
S. One capsule 2-3 times daily

No. 133

Rp. Benzoli chem. puri
Ol. Persicorum āā 0.5
D.t.d. N. 6 in caps. gelat.
S. One capsule 2-3 times daily

No. 134

Rp. Sol. Natrii nucleinici 5% 1.0
in amp. steril.
D.S. For intramuscular injection

No. 135

Rp. Sol. Thesani 0.5% 1.0
DS. 1-2 ml intramuscularly
2-3 times daily

No. 135*a*

Rp. Thesani 0.01
D.t.d. N. 12 in tabul.
S. One tablet 3 times daily

No. 136

Rp. Ferri reducti 1.0
D.t.d. N. 12
S. One powder 3 times daily,
to be washed down with dilute
hydrochloric acid

No. 137

Rp. Pil. Blaudi 100.0
DS. 1-5 pills 3 times daily

No. 137*a*

Rp. Pentoxyli 0.2
D.t.d. in capsul. gelat.
S. 1-2 capsules 2-3 times daily

No. 138

Rp. Ferri lactici 1.0
D.t.d. N. 12
S. One powder 3 times daily

No. 138*a*

Rp. Myelosani (Mylerani)
D.t.d. N. 12 in tabul.
S. One tablet 2-3 times daily

No. 139

Rp. Embichini 0.01
D.t.d. N. 10 in amp.
S. Before use take 10.0 of sterile
physiologic solution into ampule in
dilution rate indicated and inject
2-3 ml of solution

No. 139*a*

Rp. Mercaptopurini 0.05
D.t.d. N. 20 in tabul.
S. One tablet 2-3 times daily

No. 140

Rp. Campoloni 2.0
D.t.d. N. 10 in amp.
S. 2 ml intramuscularly every other day

No. 140*a*

Rp. Dopani 0.002
D.t.d. N. 6 in tabul.
S. Two tablets at 4-5-day intervals

No. 141

Rp. Antianaemini 2.0
D.t.d. N. 6 in amp.
S. 1 ml intramuscularly daily

No. 142

Rp. Sol. Natrii arsenici 1% 1.0
D.t.d. N. 10 in amp.
S. Subcutaneous injection.
Increase the dose gradually
day by day from 2 to 10
graduations; during the following
20 days the dose is of 10
graduations. After which
reduce the dose daily to reach
2 graduations

VIII. Antibiotics, Vitamins, Hormones and Chemical Drugs

No. 143

Rp. Sulfazini 1.0
D.t.d. N. 20
S. One powder 4-8 times daily

No. 144

Rp. Norsulfazoli 1.0
D.t.d. N. 20 in tabul.
S. One tablet 4-8 times daily

No. 145

Rp. Sulfadimezini 1.0
D.t.d. N. 20
S. One powder 6 times daily

No. 146

Rp. Phthalasoli 1.0
D.t.d. N. 20 in tabul.
S. One tablet 4-8 times daily

No. 147

Rp. Sulcymidi 1.0
D.t.d. N. 20
S. One powder 4-8 times daily

No. 148

Rp. Sulgini 0.5
D.t.d. N. 20 in tabul.
S. 1-2 tablets 6-8 times daily

No. 149

Rp. Penicillini 100,000 (200,000) U
D.t.d. N. 10 in amp.
S. Dissolve in physiologic solution,
100,000 U intramuscularly
at 3 hour intervals

No. 150

Rp. Biomycini hydrochlorici 100,000 U
D.t.d. N. 12 in tabul.
S. 1-2 tablets 3-4 times daily

No. 151

Rp. Streptomycini sulfurici 1.0
D.t.d. N. 10 in amp.
S. Dissolve in 10 ml of sterile
physiologic solution and
inject intramuscularly in the
morning and in the evening
in 0.5 doses

No. 152

Rp. Synthomycini 0.5
D.t.d. N. 10 in tabul.
S. One tablet 4-6 times daily

No. 153

Rp. Laevomycetini 0.5
D.t.d. N. 10 in tabul.
S. One tablet 4-6 times daily

No. 154

Rp. Acidi ascorbinici 0.1
D.t.d. N. 20
S. One powder 3 times daily

No. 155

Rp. Acidi nicotinici 0.025 (0.05)
Sacchari 0.2
M.f. pulv. D.t.d. N. 10
S. One powder 3 times daily

No. 156

Rp. Sol. Thiamini bromati 5% 1.0
D.t.d. N. 10 in amp.
S. One ampule subcutaneously

No. 157

Rp. Sol. Acidi ascorbinici 5% 1.0
D.t.d. N. 6 in amp.
S. 1 ml intravenously

No. 157*a*

Rp. Pyridoxini 0.025
Sacchari albi 0.3
M.f.p. D.t.d. N. 12
S. One powder 3 times daily

No. 157*b*

Rp. Sol. Pyridoxyni 2.5% 1.0
D.t.d. N. 10 in amp.
S. 1 ml 1-2 times daily intramuscularly

No. 158

Rp. Riboflavini 0.02
Sacchari 0.3
M.f. pulv. D.t.d. N. 10
S. One powder 3 times daily

No. 159

Rp. Vikasoli 0.05
(Vitamin K)
D.t.d. N. 20 in tabul.
S. One tablet 3 times daily

No. 159*a*

Rp. Sol. Vikasoli 1% 1.0
D.t.d. N. 6 in amp.
S. One ml intramuscularly

No. 160

Rp. Ol. Jecoris Aselli 200.0
DS. One tablespoonful 2 times daily

No. 161

Rp. Methylthiouracili 0.2
D.t.d. N. 10
S. One powder 3 times daily

No. 162

Rp. Thyreoidini 0.1
D.t.d. N. 12 in tabul.
S. One tablet 3 times daily

No. 163

Rp. Parathyreocrini 0.1
D.t.d. N. 12
S. One powder 3 times daily

No. 164

Rp. Sol. Desoxycorticosteroni acetici
oleosae 0.5% 1.0
D.t.d. N. 10 in amp.
S. One ampule intramuscularly

No. 165

Rp. Carotini 1.0
D.t.d. N. 10 in amp.
S. For subcutaneous injection

No. 166

Rp. Adiurecrini 0.1
D.t.d. N. 10
S. Inhale 2-3 times daily

No. 167

Rp. Synoestroli 0.001
D.t.d. N. 10 in tabul.
S. One tablet 2 times daily

No. 168

Rp. Sol. Folliculini 1.0 (5,000 U)
D.t.d. N. 20 in amp.
S. One ampule daily intramuscularly

No. 168*a*

Rp. Prednisoloni 0.005
D.t.d. N. 60 in tabul.
S. One tablet 4-1 times daily
according to prescription

No. 169

Rp. Sol. Emetini hydrochlorici 2% 2.0
D.t.d. N. 12 in amp.
S. 1.5 ml 2 times daily
subcutaneously

No. 170

Rp. Hormoni adrenocorticotropini
in flaconis origin 20 U
D.t.d. N. 20
S. Dissolve vial contents
in 2 ml of physiologic solution,
inject 0.5 ml intramuscularly
4 times daily

No. 171

Rp. Ovarini 1.0
D.t.d. N. 10 in amp.
S. For subcutaneous injection

No. 172

Rp. Methyltestosteroni 0.005
D.t.d. N. 20 in tabul.
DS. One tablet 2-3 times daily
(to be kept in mouth until absorption)

IX. Miscellaneous

No. 173

Rp. Dimedroli 0.03
Sacchari 0.2
M.f. pulv. D.t.d. N. 6
S. One powder 3 times daily

No. 174

Rp. Jodi puri 0.04
Kalii jodati 0.4
Extr. Valerianae q.s.
M.f. pil. N. 40
DS. One pill 3 times daily

No. 175

Rp. Jodi puri 0.04
Kalii jodati 0.4
Extr. Valerianae q.s.
Luminali 0.4
M.f. pil. N. 40
DS. One pill 3 times daily

No. 176

Rp. Jodi puri 0.03
Kalii jodati 0.3
Aq. destill. 30.0
MDS. Eight drops 3 times daily in milk

No. 176*a*

Rp. Dijodthyrosini 0.05
D.t.d. N. 40 in tabul.
S. One tablet 2 times daily after meals

No. 177

Rp. Acidi salicylici 5.0
Vaselini
Lanolini āā 50.0
M.f. ung.
DS. External

No. 177*a*

Rp. Mercasolili 0.005
D.t.d. N. 20 in tabul.
S. One tablet 2-3 times daily

No. 178

Rp. Ol. Hyoscyami
Methulii salicylici Chloroformii āā 15.0
M.f. ung.
DS. External

No. 179

Rp. Antidoti arsenici 300.0
DS. To be taken per os in 1 tablespoonful at 5-10 minute intervals

No. 180

Rp. Antidotum metallorum 200.0
DS. 50-100 ml per os

No. 181

Rp. Extr. Belladonnae 0.015
Saloli 0.3
M.f. pulv. D.t.d. N. 12
S. One powder 3 times daily

No. 182

Rp. Sol. Argenti nitrici 0.1 : 200.0
DS. One tablespoonful 3 times daily before meals

No. 183

Rp. Bismuthi subnitrici 1.0
D.t.d. N. 12
S. One powder 4-6 times daily

No. 184

Rp. Anaesthesini 1.0
Vaselini 20.0
M.f. ung.
DS. External

No. 185

Rp. Sol. Hyposulfiti natrii 10% 10.0
Sterilisetur!
DS. 10 ml intravenously

No. 186

Rp. Sol. Chinini muriatici 5% 10.0
DS. 5-10 ml intravenously

No. 187

Rp. Saponis viridis 30.0
DS. External

No. 188

Rp. Bijochinoli 30.0
DS. 2 ml every other day intramuscularly

No. 189

Rp. Novarsenoli 0.15-0.3
D.t.d. N. 6 in amp.
S. For intravenous injections; dilute with sterile water

No. 190

Rp. Sol. Kalii jodati 10.0 : 200.0
DS. 20 drops 3 times daily

No. 191

Rp. Acrichini 0.1
D.t.d. N. 12 in tabul.
S. One tablet 3 times daily

No. 192

Rp. Natrii paraaminosalicylici 3.0
D.t.d. N. 40
S. One powder 4 times daily

No. 193

Rp. Phthivazidi 0.5
D.t.d. N. 25
S. One powder 2 times daily

No. 194

Rp. Methionini 0.25
D.t.d. N. 100 in tabul.
S. 1-2 tablets 3 times daily

No. 195

Rp. Linetholi 200.0
S. One tablespoonful
2 times daily

No. 196

Rp. Khellini 0.02
D.t.d. N. 12 in tabul.
S. 1-2 tablets 3 times daily

Supplement

SPECIMEN CASE HISTORY

The patient's principal complaints upon admission to hospital

The history of the present disease (*anamnesis morbi*). This section contains a detailed description of the course and development of the disease from its first manifestations to the time of admission to the hospital. It is also necessary to indicate earlier treatment at various medical institutions and its efficacy.

The history of the patient's life (*anamnesis vitae*). Brief biographical information in chronologic order, from birth to the time of admission to the hospital: place of birth and family into which he was born, development in infancy, childhood, beginning of schooling, beginning of work and further record of the patient's employment. Sexual intercourse, menstruations, marriage, pregnancy, labour, abortion. A past history of diseases and intoxications.

Working and living conditions in the recent period: (a) working conditions: specific occupation, hygienic and working regimen; (b) housing conditions; (c) nutrition: quantity, quality, and regularity of meals.

Family history and heredity.

The Patient's Present Condition (status praesens)

General condition. Consciousness, posture. Facial expression.

Body build, height, weight.

Body temperature. Chills.

The condition of the skin: pruritus, perspiration. Colour, pigmentation, elasticity, eruptions, scars, scratchings.

Subcutaneous cellular tissue: development, oedema.

Lymph nodes.

Bones: pain, tenderness on palpation, deformities.

Joints: pains, limitation of motion, redness, shape, swelling, tenderness on palpation, active and passive movements.

The respiratory system. Nasal respiration, nasal discharge. Nosebleed. Chest pains. Dyspnoea, asthmatic attacks. Cough, sputum, haemoptysis.

Thorax. Form, symmetry, deformities, circumference.

Respiration. Type, symmetry of respiratory movements, depth and rhythm of respiration. Number of respirations per minute. Palpation of the thorax.

Comparative and topographic percussion, upper and lower lung boundaries, excursion of the inferior lung edge (in centimetres along the midclavicular, midaxillary and scapular lines).

Auscultation. Character of respiration, rales, pleural friction rub, bronchophony.

The cardiovascular system. Palpitation, irregular heart beats, pains and other unpleasant sensations in the heart region. Bulging and pulsation in the region of the heart and larger vessels. The cardiac and apical thrust: location and character. Purring thrill. Epigastric pulsation; pulsation of the liver. Boundaries of the heart: right, upper, left; width and configuration of cardiac dullness; width of vascular dullness in the second intercostal space.

Auscultation of the heart and larger vessels: sounds, murmurs.

The pulse in the radial arteries: its contour, frequency, rhythm, tension, rate, volume.

Arteries: elasticity, tortuousness, pulsation, auscultation of arteries. Engorgement and pulsation of the cervical and other veins. Varicose dilation of veins. Blood pressure (arterial and venous).

The digestive system. Appetite, thirst, mastication, deglutition, passage of food through the oesophagus, eructation, heartburn, nausea, vomiting, pain and other abnormal sensations in the epigastrium, right and left hypochondrium, and other parts of the abdomen.

Intestinal function, haemorrhoids.

The lips, gums, teeth, tongue, fauces, tonsils, pharynx.

Abdomen: shape, visible peristalsis, tone of abdominal muscles, circumference of the abdomen, ascites. Palpation of the stomach and intestine. Digital examination of the rectum.

The liver. Upper and lower boundaries and diameter along the right midclavicular line, lower boundary along the median line, palpation (margin, surface, tenderness).

The spleen. Size, findings on palpation.

Urogenital system. Micturition in the daytime, at night, symptoms of dysuria, diuresis, pains in the kidney region. Pasternatsky's symptom, palpation of the kidneys. Scrotum, testes, prostate. Menstruations, pains, leucorrhoea.

Neuropsychic condition. Consciousness: clear, confused, dimmed. Mood: balanced, calm, excited, depressed, anxious, aboulia, etc. Fixed ideas and delirious ideas and their character.

Sleep: normal, disturbed (character of disturbance).

Headaches: character, a buzzing sensation in the head, dizziness, meningeal symptoms.

Hallucinations: acoustic, visual, olfactory.

Cranial nerves.

Tendon and periosteal reflexes.

Skin reflexes (abdominal, cremasteric).

Reflexes from the mucous membranes (conjunctival, pharyngeal).

The motor system and coordination of movements (paralysis, paresis, muscle strength and tone, gait, station).

Sensory functions.

Dermographia.

Pathologic reflexes.

Vision, eyeball. Exophthalmos, enophthalmos, nystagmus, pupils, pupillary reflexes.

Hearing.

Findings in laboratory, instrumental and special examinations. (Results of laboratory tests are recorded in a special card; data of instrumental and special examinations are registered in the patient's medical record as soon as they are collected.)

Examination of sputum. Examination of pleural, ascitic, and cerebrospinal fluids. Examination of the blood: morphologic, biochemical, bacteriologic, and serologic. Examination of gastric contents. Examination of duodenal contents. Examination of faeces. Urinalysis. Examination of kidney function (Zimnitsky's and other tests).

Roentgenoscopy of the thoracic organs, oesophagus, stomach, intestine, gallbladder, kidneys, ureters, and urinary bladder. Roentgenography of the bones and joints and other roentgenological studies. Electrocardiography. Bronchoscopy, oesophagoscopy, gastroscopy, rectoromanoscopy.

Examination of the ear, nose and throat. Examination of the organ of vision, fundus oculi. Gynaecologic examination. Urologic examination.

Other laboratory and instrumental examinations not listed above, as well as consultations are undertaken whenever required and their findings are recorded in a card for laboratory tests or in the medical record.

Diagnosis of the disease. Medical record. *Epicrisis* (brief substantiation of diagnosis, characteristics and specific features of the course of the disease in the given patient, treatment carried out, the patient's condition upon discharge. The prognosis of survival, rehabilitation, working capacity; further measures required, choice of the patient's occupation in particular).

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TO THE READER

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